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AMERICAN ASSOCIATION OF NURSE ANESTHETISTS

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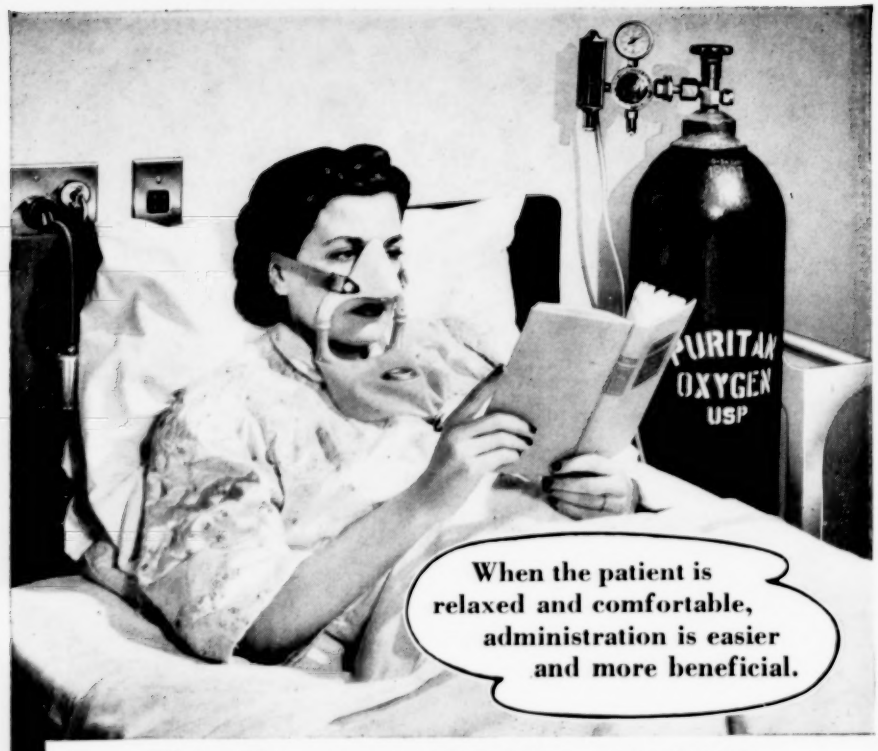

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1. Goodman, L., and Gilman, A.: *The Pharmacological Basis of Therapeutics*, New York, The Macmillan Co., 1947, p. 58.

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OPINION REVIEW

PRACTICAL NURSES AND POSTANESTHESIA CARE

Owing to the shortage of registered nurses, the practical nurse is a must in our hospital. We use them in every department except surgery and the labor and delivery rooms.

There is a central training school here in Fort Worth for practical nurses. The course lasts a year. To enroll, the student must have had two years of high school and be between the ages of 17-50 and in good health. For the first three months of the course theoretical instruction is given at the school. Then the student practical nurse does nine months' work in one of the hospitals where she gets her practical training under the supervision of graduate nurses. At the same time, she receives four hours of related class work per week at the school. At the end of the year, if the student has proved satisfactory, she becomes a graduate practical nurse. As yet, the practical nurse is not licensed in Texas.

As I understand it, the school was established with the idea of providing personnel for the care of the convalescent and the chronically, but not seriously, ill patient. Should it become necessary to use the practical nurse in the care of the acutely ill patient, she must have graduate nurse supervision. Here at All Saints Hospital, there is graduate nurse supervision whether the patient is convalescing or acutely ill, and whether the practical nurse is a student or graduate.

In relation to the postanesthesia patient, the practical nurse is taught how to take blood pressure, to recognize the different types of pulse and respiration, to recognize pallor and cyanosis, what to do if the patient vomits, to watch intravenous fluids, to watch for bleeding, diaphoresis, catheter drainage, restlessness, and so forth. Any deviation from what she has been taught as normal is immediately reported to the graduate nurse floor supervisor, who is in close attendance, and who is in the room as often as possible to check the patient's condition. The supervisor institutes the necessary treatment.

The majority of the practical nurses are mature individuals who have already reared families, and who are used to taking responsibility. Rarely is age a factor against them. Occasionally, an older woman appears less adaptable than her younger sister. And it seems that occasionally one of them feels that her maturity makes her judgment better than that of a young graduate nursing supervisor. But most of them are keenly interested, alert, proud of the usefulness of their jobs, and dependable. On the whole, the practical nurse is satisfactory working under the supervision of the graduate nurse.—Alva West, R. N., All Saints Hospital, Fort Worth, Tex.

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*Excerpt from Memorial Address at 1936 Clinical Congress of Surgeons.

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My opinion as to the advisability of the use of practical nurses in postanesthesia recovery departments is that there are many and varied duties which they may be trained to do efficiently under direct supervision of a graduate nurse.

I do not feel that they should be allowed to accept the responsibility of making decisions concerning the patients' care, but should work as assistants to the nurse in charge.—MARY A. SHAFNER, R. N., St. Joseph's Hospital, Fort Worth, Tex.

The consensus around the Pittsburgh area in regard to the use of practical nurses in the postanesthesia care of the unconscious patient is an emphatic "no."

Upon quizzing several staff doctors in this hospital about their opinions, I received these answers: They did not care as long as a graduate nurse was handy in case the practical nurse summoned her. Or they relied on the judicious judgment of the floor supervisor to determine the capability of the individual practical nurse.

Upon questioning some of the floor supervisors in regard to their "judicious judgment" and the capabilities of the individual practical nurse, I received this reply:

In a group of practical nurses there is one or perhaps several girls who have had some education and practical experience in a school of nursing, but who have not received a nurse's diploma and because of unknown circumstances have accepted practical nursing as an alternative. These are the individuals who know how to manage an unconscious patient for postanesthesia care and are dependable in carrying out any instructions.

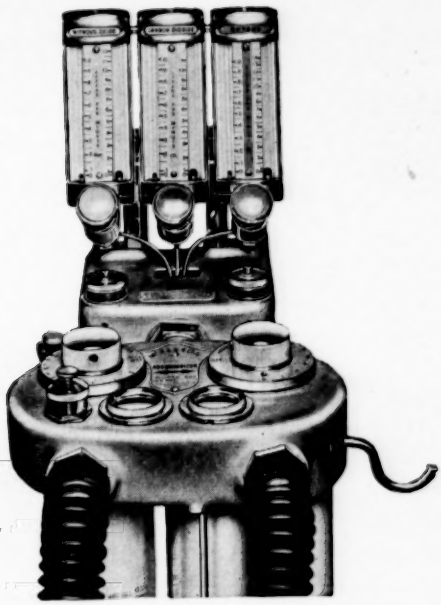
Another instance when a practical nurse may be used for this particular reason is in a postoperative ward where graduate nurses are on duty all the time.—MATHILDA M. MARGISON, R.N., Sewickley Valley Hospital, Sewickley, Pa.

Our nurses' aides are taught and supervised by an instructor in the hospital and are then sent to the recovery room where they are under constant supervision. We consider these aides a most essential part of this department, as they are capable of doing routine duties; this enables the graduate nurse to be available for those patients requiring special attention owing to the more serious complications as a result of surgery.

The aides for the recovery rooms are chosen very carefully as to their intelligence, dependability, and ability to work into such a picture. These people are capable of staying with anesthetized patients, as they have been trained what things they can do relative to this type of patient, and their supervisor is always within close range to be of assistance.

Our surgical recovery department at Harris Hospital opened in May of 1944. Since the opening of this department we have had an auxiliary nurse or nurse's aide on each eight hour shift.

The duties of the nurse's aide are to: (1) keep the department in order and clean and take care of equipment; (2) check supplies in general, both in receiving them from and returning them to central supply room; (3) help to admit the patients to surgical recovery for postanesthesia care: (a) apply T-binders, (b) give hygienic care, (c) apply pressure cuff for blood pressure, (d) notice general condition; (4) assist in turning patients; (5) give arm and leg exercises; and (6) transfer patients to rooms when they are able to leave the recovery rooms.—R. N., Harris Hospital, Fort Worth, Tex.



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"Pay Your Money and . . ."

Some of the reports of poor attendance at spring meetings remind us of the woman who spent two hours selecting a spring hat, paid for it, went off without it, and never did go back to pick it up. Whatever her trouble was, it is sufficiently uncommon among shoppers to elevate eyebrows. Yet when a parallel in foolishness crops up in an association, it seems to provoke no reaction of surprise whatsoever. Evidently there is nothing peculiar about the person who will spend years in preparation to become eligible for membership in a professional organization, pay dues without a murmur to maintain that affiliation, and then walk off without picking up the benefits.

Innumerable excuses are offered for a member's failure to get his money's worth from an organization. "I can't afford to attend meetings." "They won't give me time off from the hospital." "What difference would it make; the same old crowd will run things anyway." But who can afford *not* to protect a capital investment, such as a nurse makes when she spends time and money to qualify as an anesthetist? Who would say that it is "impossible" to get time off from any job when that time is to be spent in the interest of the employer? And who would argue that the crowd that runs things should turn over responsibility to persons who have sought no information and shown no interest?

Although it is admittedly difficult for every anesthetist to get time off to attend every meeting, if the anesthetists are interested, schedules can be adjusted to allow the hospital to be represented at both state and national conventions.

All too often the excuse-makers are also the complainers when an association does not function as they think it should. They cry afterwards about policies made, officers elected, and issues discussed at meetings they never attended.

The dues-paying non-participant, in a sense, puts the professional organization in the position of extracting tribute, a practice that has little to recommend it except to feudal societies and gangs of "hoods." Associations should and *do* offer more than "protection." And when you join an association, you're doing more than buying a hat.

THE CLINICAL USE OF CURARE IN ANESTHESIA

Opal M. Schram, R.N.*
Chicago

Since 1942, when curare was introduced into anesthesia, probably every anesthesia publication has contained one or more articles on the drug and the manner of its use. Why then another article? It has been pointed out that this considerable volume of literature is not available to many nurse anesthetists who are called upon to give the drug, and that these anesthetists wish to know the points that are essential to its safe and effective use. Reduced to a minimum, the information necessary includes: (1) the action of curare, (2) its application to anesthesia, (3) the method of use, (4) preparations available, and (5) the dangers that may be encountered.

Action of Curare

Curare is an extract from a variety of plants and has been popularly known as an arrow poison used by South American Indians. Death is produced by asphyxia due to paralysis of the muscles of respiration. The drug has a very interesting history,[1] the perusal of which is well worth a portion of the leisure time of any anesthetist.

Curare is not an anesthetic agent, having neither the ability to produce sleep nor to obtund pain. It is useful in anesthesia only because it produces relaxation, chiefly of the skeletal muscles. It produces this effect by blocking the nerve impulse at its junction with the muscle, thus rendering the muscle flaccid. Curare has a selective action, affecting first the muscles of the head and the neck, then those of the extremities and the abdomen, then the intercostal muscles, and finally the diaphragm. As the drug is excreted, recovery takes place in the reverse order. When the drug is given in therapeutic doses, the action is believed to be entirely peripheral; the reduction in the respiratory exchange is due to muscle paralysis and not to depression of the respiratory center, and there is no direct effect on the heart or circulation. In marked overdosage of the drug there may be both circulatory depression and central depression of respiration. Curare is believed to be partially destroyed in the liver and partly eliminated unchanged by the kidneys. However, it has not been shown that liver or kidney impairment prolongs or intensifies the action.[2]

Application to Anesthesia

It is well known that a light plane of anesthesia is better tolerated by a patient than a deep plane, and that a light plane is followed by fewer toxic effects. It is also well known

*Director, School of Anesthesia for Nurses, Wesley Memorial Hospital.

1. Gill, R. G.: *White Water and Black Magic* (New York: Henry Holt & Company, 1940).

2. Cullen, S. C.: Clinical and laboratory observations on the use of curare during inhalation anesthesia. Anesthesiology 5:166-173, March 1944.

that in any case a large portion of the anesthetic drug used is administered to insure a quiet, relaxed patient. Obviously, then, a drug that will produce quiet and relaxation in a light plane of anesthesia has much to recommend its use.

METHOD OF USE

Curare is effective almost immediately when administered intravenously, and in fifteen or twenty minutes when given intramuscularly. It is without effect when administered orally. The intravenous route of administration is the most readily controllable in anesthesia and is preferred by most anesthetists. It is desirable to have an infusion of fluids started intravenously before the operation is begun so that the anesthetist can give the curare at the time she wishes. Some surgeons object to the use of ankle veins, some to arm boards, and others to both. In the last instance it is quite possible, when a large needle is used, to start the administration of fluids in a vein of the forearm or hand, and then to put the arm at the side and tuck it in with a draw sheet in such a manner that the fluid will continue to drip. This is a purely mechanical matter, but it assumes considerable importance if you have planned to secure relaxation with curare, the moment of need has come, and the needle is clogged or out of the vein.

For abdominal operations the dose of curare should be sufficient to produce some paresis of intercostal muscles, but it should not be so marked that the respiration is entirely diaphragmatic, jerky, and inadequate. Experience will aid the anesthetist in estimating the dosage. She will take into consideration the size, vigor, and musculature of the patient, the site of operation, and the anesthetic agent to be used.

Generally speaking, the larger, more vigorous, and muscular the patient, the more curare will be needed to relax the muscles. Upper abdominal operations may require larger doses than lower abdominal operations, but the use of a reasonable curarizing dose, with a deeper plane of anesthesia to control the reflexes resulting from manipulations in the upper abdomen, may be a safer and perhaps more rational method. In considering the relationship of the anesthetic agent to the dosage of curare, it must be remembered that nitrous oxide, ethylene, and cyclopropane have no effect on the action of curare. In pentothal sodium anesthesia the action of curare may be a little more profound than in anesthesia with the gaseous agents. Ether has very definite curariform properties, both increasing and prolonging the curare action. It is therefore advised that the dose of curare be cut in half or even further reduced, depending upon the plane of ether anesthesia. Avertin also appears to have some curariform action, and the dose of curare should be reduced accordingly. If the anesthetic agent is cyclopropane, 2½ to 3 cc. (50-60 units) of Intocostrin or d-tubocurarine chloride is a reasonable initial dose for an average adult patient undergoing laparotomy.

Premedication is the same as for any type of general anesthesia. Atropine or scopolamine is essential, as there is an increased tendency to mucus secretion when curare is used. A well accepted procedure is to anesthetize the patient with cyclopropane — pentothal sodium induction if preferred — to upper plane two. The eyes will be just centered, the intercostal muscles active, and the respiratory ex-

change good. About five minutes before the peritoneum is to be opened, the estimated dose of curare is given intravenously. If the estimated dose is about right, within a few seconds to a few minutes a definite change in respiration is evident. The movement of the breathing bag will show reduced exchange, and observation of the patient will show the breathing to be largely diaphragmatic. The abdominal wall will be flaccid and the bowel contracted and quiet. The pulse and blood pressure should not be affected materially. If there is an increase in blood pressure due to reduced respiratory exchange, the respiration should be augmented by gentle manual pressure on the breathing bag at the beginning of inspiration. Often it is sufficient to assist every other inspiration; in this manner it is easier to work with the patient, and there is less likelihood of getting into controlled respiration. It is desirable to avoid this degree of respiratory reduction, but if it occurs, it is much better to augment the respiration than to permit oxygen want and carbon dioxide excess. The newer, methylated curare preparations* appear to be capable of producing satisfactory relaxation without significant respiratory depression.

If relaxation is not satisfactory, the anesthetist must decide whether it is the plane of anesthesia or the degree of curarization that is inadequate. If the anesthesia seems satisfactory and some activity of intercostal muscles is present, the patient will probably tolerate more curare. The amount will depend upon the effect already produced. Cullen[3] suggested one third to two thirds of the initial dose. Five minutes should be allowed for the full effect of each dose.

If it is decided to increase the concentration of the anesthetic agent and it is desired to use some ether, to avoid either anoxia or the unfavorable cardiac effects of cyclopropane, it may be added cautiously after the dose of curare has reached its maximal effect. This practice is not attended by the same danger as the administration of curare to a patient under ether anesthesia. Ether is slow in action, and if the effects are watched, its administration can be stopped at the proper time; also some desaturation can be achieved if necessary. On the other hand, when ether saturation is present and curare is given, the effect will be sudden and practically irreversible. Probably no one can unfailingly estimate the dose of curare that will produce just the right amount of muscular paralysis. The presence of ether in the circulating blood adds another unknown to the individual variability that must always be dealt with.

If the dose of curare has been too large, marked respiratory reduction or arrest will occur. This condition is generally not at all serious, but it must be handled by immediate efficient controlled respiration. The anesthetist must be certain that she is providing a clear exchange of gases, and that the concentration of the anesthetic agent is such that it will not add to the depression or permit the patient to awaken. The condition may last from five to twenty minutes, but if pulmonary ventilation is adequate, the pulse and blood pressure will remain stable, and the patient will suffer no ill effect. Sometimes, when the dose is too large, the respiration will become jerky, and tracheal tug will occur.

*Halogen salts of d-tubocurarine dimethyl ether.

3. Cullen, S. C.: The use of curare for the improvement of abdominal relaxation during inhalation anesthesia. Anesthesiology **14**:261-266, Aug. 1943.

Such respiration should be augmented, both to provide more adequate oxygenation and to improve relaxation in the operative field.

The length of action of curare varies with the individual patient, the anesthetic agent, and the curare preparation used. D-tubocurarine chloride will disappear from the blood stream in about twenty minutes, but the relaxing effect will usually last forty-five minutes to one hour after administration. The methylated curare preparations seem to act as long as one and one half to two hours. It is well to watch for the return of activity of intercostal muscles and then for return of tone in the abdominal muscles. Doses required to maintain relaxation will usually be a third or less of the original dose. If possible, activity of intercostal muscles should be present when the patient is ready to leave the operating room.

Curare may be used for relaxation with any anesthetic agent or combination of agents if consideration is given to any curare-like action of the anesthetic drug used. The pentothal sodium-curare-nitrous oxide combination is very popular with some anesthetists.[4] According to Cullen,[4a] when curare is used with nitrous oxide or ethylene, "all the relaxation must be obtained with the curare, and the hazards attending its excessive and prolonged use appear." If ether is added in small amounts to these gases, this difficulty is overcome, and when experience is acquired in estimating the dosage of curare, the combination can be quite satisfactory. Cyclopropane-curare is used extensively, and fewer cardiac irregularities are encountered with the combination because of the lower concentration of cyclopropane needed. However, postanesthesia circulatory depression (so-called cyclopropane shock) is just as likely to occur as if no curare were used. It would seem that the beginner in the use of curare would be well advised to use the anesthetic agent with which she is the most familiar, and to calculate the amount of curare according to the curariform properties of the agent used.

4. Knight, R. T.: Combined use of sodium pentothal, Intocostrin (curare) nitrous oxide. Canadian M. A. J. **55**:356-360, 1946.

4a. Cullen, S. C.: Curare and anesthesia. Surgery **18**:45-47, July 1945.

CURARE PREPARATIONS AVAILABLE

The anesthetist who plans to use any drug should familiarize herself with the description furnished by the company manufacturing the drug. In addition, many drug companies will supply reprints from the medical literature, so no one need suffer from lack of information.

The first curare preparation available to anesthetists was Intocostrin (Squibb), which was introduced in 1942. D-tubocurarine chloride, the active principle in Intocostrin, was soon isolated, and in a short time many drug companies were offering the drug under the name d-tubocurarine chloride. These preparations seemed to have essentially the same potency per cubic centimeter of solution, although the number of milligrams varied. Most of these companies advised that the preparation be kept under refrigeration in order to retain its potency. At the present time d-tubocurarine chloride (3 mg. per cubic centimeter, 20 units per cubic centimeter) is the drug most anesthetists refer to when they mention curare.

The methylated curares (halogen salts of d-tubocurarine dimethyl ether) were referred to earlier in

this discussion. They have been under investigation for a number of years, and Metubine Iodide (Lilly) has been available since February 1949. E. R. Squibb & Sons will soon release a similar preparation called Mecostrin. Comparatively little about these preparations has yet appeared in the literature, but both have been used extensively as experimental drugs in this hospital.* In the proper dosage they produce good relaxation with definitely less respiratory effects than are produced by d-tubocurarine chloride, and the action is longer.

It was found that the number of cubic centimeters of Metubine Iodide required to produce relaxation was approximately three times that of the d-tubocurarine chloride solutions. Since Metubine Iodide solution contains 0.5 mg. of drug per cubic centimeter and the d-tubocurarine chloride solutions 3 mg. per cubic centimeter, the actual amount of drug is less. The potency of Mecostrin is twice that of Metubine Iodide per cubic centimeter of solution. This is true in spite of the fact that Metubine Iodide is labeled 0.5 mg. per cubic centimeter and Mecostrin is labeled 0.8 mg. per ion/cubic centimeter. The explanation is that Lilly records the total weight of the molecule, while Squibb records only the weight of the active ion, leaving out the weight of the inactive chloride and the water. Lilly will soon release a Metubine Iodide preparation with twice the strength of the present solution, i.e., with the same potency as Mecostrin.

During the period of experimental use of these drugs, Davis[5] devised an ingenious table for the estimation of dosage. He based the table on the body surface of the patient. The dosage determined by this scale has proved to be amazingly accurate and is relied upon by the anesthetists in the hospital where the method was studied.

Syncurine (decamethonium bromide [C 10] Burroughs Wellcome) is a new synthetic skeletal muscle relaxant. The potency per milligram of drug is estimated to be three to five times that of d-tubocurarine chloride. The solution contains 2 mg. of drug per cubic centimeter, and ½ cc. is considered a safe dose. Reports indicate that marked respiratory depression or paralysis occurs more frequently after the use of this drug than of other curare preparations. Foldes and Machaj[6] reported that satisfactory relaxation was obtained in all cases, but not without the development of apnea in one third of the cases.

The duration of action of Syncurine is less than that of the other preparations. It is estimated to be about twenty minutes. It would therefore seem to be especially indicated for electric shock therapy and perhaps in anesthesia when only brief relaxation is needed, as for peritoneal closure.

It is reported that Syncurine has no tendency to produce histamine-like reactions, such as a decrease in blood pressure and bronchospasm, which have occurred occasionally with the use of the other curare preparations.

Myanesin (one of the a-substituted ethers of glycerol) is a skeletal muscle relaxant that was introduced in 1947. British anesthesiologists reported favorably on its actions. It was quite thoroughly investigated

*Wesley Memorial Hospital, Chicago.

5. Davis, Neal: Dosage guide for the administration of the halogen salts of d-tubocurarine dimethyl ether. Indiana M. J., to be published.

6. Foldes, F. F., and Machaj, T. S.: To be published.

by Griffith and co-workers,[7] who found that its action was less predictable than that of curare, and that it occasionally caused local irritation at the site of injection and hemoglobinuria. They concluded that Myanesin has no advantage over curare and in its present form is not a satisfactory substitute.

DANGERS OF CURARE

When curare is used in a rational manner, there seem to be very few complications. It is well known that myasthenia gravis is a true contraindication to its use. But this condition is rarely undiagnosed, and the chances of such a patient's receiving curare are fortunately remote. Some anesthesiologists believe that curare should not be given to patients with bowel obstruction for fear of increasing the danger of regurgitation and aspiration. This danger is eliminated if the patient is intubated and an inflatable cuff or pack is used to isolate the trachea.

Histamine-like reactions, such as a decrease in blood pressure and bronchospasm, have been known to occur following curare administration, the latter particularly when the anesthetic agent is cyclopropane. Administration of additional curare has been suggested to overcome bronchospasm, but Dr. Mary Karp,[8] director of the Department of Anesthesia at Wesley Memorial Hospital, advises the discontinuance of both the cyclopropane and curare, and a change to ether-oxygen anesthesia, with helium and pressure if necessary to secure a good respiratory exchange. If bronchospasm is extreme, Isuprel or aminophylline may be given intravenously. The recommended dosage of Isuprel is 1 cc. (1:50,000) and of aminophylline 250-500 mg. Severe bronchospasm is a serious emergency, but its occurrence must be quite rare, as not many cases have been reported.

The commonest complication encountered with the use of curare is respiratory depression or apnea. Not even the smallest amount of curare should be given unless the anesthetist is prepared to perform immediate efficient artificial respiration. Prostigmine, or neostigmine, has been referred to as a physiologic antidote, but dependence on antidotes gives a false sense of security. Prostigmine, or neostigmine, increases mucus secretion in the respiratory tract, and if used, atropine should also be given. Cullen[9] warned against the use of these drugs and stated that prostigmine may cause or contribute to bronchospasm. When overdosage has been great enough to cause circulatory depression, prostigmine is likely to cause a further decrease in blood pressure. Most authorities recommend careful estimation of dosage and management of respiratory depression or apnea with oxygen and artificial respiration in preference to the use of antidotes. It is significant that, in eight years of use of the curare preparations in this hospital, we have found no occasion to use either prostigmine or neostigmine, nor have we seen bronchospasm of any severity.

SUMMARY

An attempt has been made to describe the action of curare and its use in anesthesia. The manner of its use with cyclopropane anesthesia

(*Continued on page* 134)

7. Griffith, H. R.; Stephen, C. R.; Cullen, W. G., and Bourne, Wesley: Myanesin as a muscle relaxant. Anesthesiology **10**:61-65, Jan. 1949.

8. Karp, Mary: Personal communication to the author.

9. Cullen, S. C.: Curare: Its past and present. Anesthesiology **8**:479-488, Sept. 1947.

CEREBRAL MANIFESTATIONS OF ANOXIA

A Review of the Literature
Part III

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In the continued search of the literature for information concerning the effects of anoxia on the cerebral cortex, some of the foreign journals were studied. Although there was evidence of an interest in the subject, the foreign literature was not so productive of pertinent data as was the English literature.

Pirondi[1] (1881) reported some secondary effects of anesthesia. The delayed effects were described as slight disturbances of the digestive system and prolonged drowsiness, lasting from three to six days. One patient who was given nitrous oxide was profoundly drowsy for three days. Other delayed effects were motor difficulties, "troubled vision," and speech disorders. The author considered these sequelae as not serious. In Pirondi's opinion sensitivity of the nervous system was among the predisposing causes of such phenomena.

Cramer[2] (1928) reported his findings after more than 10,000 administrations of ether, chloroform, or a mixture of ether and chloroform. He described the psychic disturbances that follow inhalation anesthesia. Although the physical status of the patient may be satisfactory, mental changes may develop. Decreased mental power, moodiness, loss of memory, and lessened ability to learn were among the changes noted. The duration of these mental disturbances varied from days to many months. Experiments on students confirmed the author's impressions. The author believed that scopolamine, avertin, and narcylene were worse offenders than ether or chloroform in the production of psychic disturbances. These disturbances were attributed to the direct toxic effect of the agent on the cells of the brain, and the author recommended the speeding of elimination by carbon dioxide inhalations.

Béhague and Garsaux[3] (1930), in studies of a pilot's reactions at varying altitudes, found that paralysis of one arm developed at 13,800 feet. The weakness disappeared after a day. Rabbits were subjected to comparable conditions, and paralysis of one or more legs developed. The paralyses disappeared at various times; some lasted as long as one month. All the quadriplegic animals died within forty-eight hours after the experiment. The authors attributed the paralysis to cerebral anoxia caused by diminished pressures, diminished respiratory exchange, and diminished blood flow to the brain.

Stubbe-Teglbjaerg[4] (1932), investigating the Lennox theory of

*Executive Director, A.A.N.A.; Co-editor *Anesthesia Abstracts*.

1. Pirondi, Sirus: Some secondary effects or delayed actions of anesthetics and more particularly from use of chloroform. Marseille-med. **18**:208-216, 1881.
2. Cramer, H.: Psychic disturbances produced by inhalation narcosis. Munchen. med. Wchnschr. **75**:2008-2009, Nov. 23, 1928.
3. Béhague, P., and Garsaux: Transitory paralysis after diminution of oxygen at high altitude; probable cerebral anoxemia. Rev. neurol. 1:77-79, Jan. 1930.
4. Stubbe-Teglbjaerg, H. P.: Epilepsy and hypoxemia. Acta psychiat. et neurol. 7:659-78, 1932.

hypoxemia as a cause of epilepsy, experimented on himself and fifty epileptic persons. He produced anoxia and recorded the psychologic changes, which included loss of attention, distraction, loss of control of voluntary movements, loss of memory, failure of judgment, impairment of vision and hearing, euphoria, anxiety, anger, and finally loss of consciousness. Although the experiments were conducted with the greatest caution, the author believed that a short anoxic episode, even leading to the unconscious state, produced no serious sequelae.

Goralewski[5] (1935, 1936) used fifty persons in a series of experiments designed to study the cerebral effects of anoxia. He found that varying levels of anoxia produced tremor, ataxia, and athetosis. In deeper phases of anoxia, loss of control of direction changes, speech disorders, and jerking movements developed. These reactions were varied; they were not produced at the same levels of anoxia in all persons.

Mehring[6] (1942) studied the sequelae of anoxia and listed loss of memory, amnesia of varying degrees, illusions, false judgment, moodiness, and incoordinated movements. In one case reported, a man, aged 18, was anesthetized with ether for appendectomy. Four minutes after the administration of the anesthetic was started, 12 to 15 cc. of ether having been used, respirations ceased. Artificial respiration restored normal breathing, and cyanosis disappeared. The patient did not arouse for twenty-four hours, although coramine, blood transfusion, and other measures were tried. Upon arousing he was amnesic and had an increase in temperature to 40.5 C. On the fifth day he became excited, gay, and noisy. Nystagmus, speech difficulty, and tremor of the fingers were noted. Contrary to his former habit, he manifested a desire for liquor. Thirty-one days after the operation, his only complaint was tremor of both legs.

In the English literature, continuing interest in the subject of cerebral manifestations of anoxia is apparent. Among the recent articles are two that may be of special interest.

Keith and Norval[7] (1950), in a long term follow-up of patients, reported that, in a series of 111 babies who experienced asphyxia or delayed respiration or both, there were more deaths and more abnormal infants than among the controls. If the babies survived the neonatal period, their development was normal, "or at least no more abnormalities were noted among the asphyctic infants than among the controls." The authors concluded that "Anoxia caused by delay in the onset of respiration did not have a serious prognosis in those infants who survived the neonatal period. Even a delay of 11 to 15 minutes, while fatal in 2 instances, did not appear to cause any abnormality, at least during the first four years of life, in the infants who survived."

A phase of the problem of postanesthesia encephalopathy that had not previously been investigated was presented by Seldon et al[8] (1949). Two cases were presented to sug-

5. Goralewski, G.: Disturbances of central nervous system in anoxemia. Arbeitsphysiol. **9**:94-118, 1935; Disturbances of central nervous system (extrapyramidal) in anoxemia. Arbeitsphysiol. **9**:392-413, 1936.

6. Mehring, C. E.: Lesions of central nervous system (complications and sequels of anesthesia). Deutsche mil.-arztl. Ztschr. 7:640-644, Oct. 1942.

7. Keith, H. M., and Norval, Mildred A.: Neurologic lesions in the newborn. Proc. Staff Meet., Mayo Clin. **25**:11-13, Jan. 4, 1950.

8. Seldon, T. H.; Faulconer, Albert, Jr.; Courtin, R. F., and Pino, D. M.: Postanesthetic encephalopathy: The postulation of cerebral edema as a basis for rational treatment. Proc. Staff Meet., Mayo Clin. **24**:370-374, July 6, 1949.

gest the role of edema and to offer a method of treatment.

The first patient, a woman, aged 27, was to have a right radical mastectomy. There was no record of difficulty after four previous operations. Nitrous oxide-oxygen and ether were given. An intratracheal tube was inserted to relieve moderate phonation. Semi-open drop ether was used after the breast was removed. Five ounces of ether was used during two and one-half hours of anesthesia. At no time during the procedure was cyanosis or any other abnormal sign noted.

Two hours after the completion of the operation, the patient mumbled when stimulated and appeared to be awakening. Twelve hours later she was comatose. During the next few hours she seemed to attempt to answer questions. Desoxyn (desoxyephedrine), 5 mg., was given intravenously to counteract the cortical depression. A generalized convulsion occurred after this medication was given. This episode and two succeeding episodes of convulsions were controlled by intravenous injection of a few cubic centimeters of pentothal sodium. After consultation with a neurologist it was decided to treat the patient for cerebral edema. Twenty-five hours after the operation was completed, 100 cc. of 25 per cent human serum albumin was given. Improvement continued, and four days after operation the patient was fully conscious. The results of neurologic examination on the twenty-fifth postoperative day seemed to be within normal limits.

The second case was of a man, aged 50, who was to have an operation for carcinoma of the tongue. Because of a recent dissection of cervical nodes and ligation of arteries, and because of the extent of the lesion, there was some obstruction of the airway. Pentothal sodium was used for induction of anesthesia. After 750 mg. pentothal sodium had been administered in two minutes, complete obstruction of the airway resulted from laryngospasm. Cyanosis became pronounced. A laryngoscope was passed, but the larynx could not be visualized. A tracheotomy was done. It was possible to pass an intratracheal tube just before the tracheotomy was completed. Eight minutes had elapsed from the time laryngospasm had started.

One hour and fifteen minutes after induction of anesthesia had been started, the patient moved his extremities in a purposeless manner; the postanesthesia period was in contrast with the usual quiet recovery after pentothal sodium anesthesia. After examination four hours later, the neurologist suggested that the patient had postanoxic cerebral edema. Three hundred cubic centimeters of 25 per cent human serum albumin was given in 100 cc. doses at four hour intervals. Five minutes after the first dose, improvement was noted. By the time the second injection was given, the patient showed signs of almost normal cerebration. By the time the third injection was given, he was alert and fully cooperative.

In commenting on the method of treatment, the authors said:

> Inasmuch as human serum albumin is known to be a most effective agent for the reduction of cerebral edema, the conjecture cannot be avoided in the cases presented that the improvement observed was a result of coincident improvement in the cerebral edema following the anoxic episodes.
>
> We suggest, therefore, the following revision of our time-honored views re-

(*Continued on page* 97)

MANAGEMENT OF CERTAIN COMPLICATIONS THAT OCCUR DURING ANESTHESIA

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I approach with much diffidence the subject that has been assigned me; from the literature, I am fully aware of the considerable number of complications that arise during anesthesia in even the foremost institutions of this country, as well as in the most advanced in foreign lands. While a complete coverage of the subject would be far beyond the scope of any one brief article, a résumé of my own and of others' experience in preventing or correcting certain complications that, from the literature, obviously occur many times under varying circumstances may prove of interest.

CONVULSIONS

Among the most commonly met complications are the so-called *convulsions* that occur during anesthesia. While these were described as "ether convulsions" in the literature of some sixteen years ago, when the anesthetic in general use was ether, experience with later technics of anesthesia has revealed the fact that these convulsive seizures occur when other anesthetic agents as well are used.

To provide a background for evaluating the complication, I will briefly review some of the early literature on the subject. In 1932 Daly[1] of England reported the case of a patient in whom, after thirty minutes of anesthesia, during closure of the peritoneum, convulsions developed in the face and jaw and spread to the rest of the body. Administration of the anesthetic was discontinued, and carbon dioxide and oxygen were administered to hasten the elimination of ether; but the convulsions continued, and closure of the abdomen was effected with great difficulty between the spasms. Because the patient's face was flushed and congested, the head of the table was raised with the object of relieving the congestion. The convulsions ceased at once and did not recur.

That same year, Blomfield[2] of England published an appeal for information concerning the cause and correction of "generalized" convulsions during ether anesthesia, which he described thus: "The attack generally begins with twitchings of the face muscles, when the patient is deeply under the anesthetic. The twitchings are followed by spasmodic contractions of the limbs and of most of the muscles of the body—leading to cessation of the operation, and in many instances ending fatally either on the operating table or later. While some of

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*Director, School of Anesthesia, Barnes Hospital.

1. Daly, A.: Ether convulsions: A note as to treatment. Brit. J. Anaesth. **9**:67-68, Jan. 1932.

2. Blomfield, J.: Convulsions during ether anesthesia; Wanted, information. Anesth. & Analg. (supp) 11:38, May-June, 1932.

the patients have been suffering from acute infection, others have been in normal health. These attacks must not be confused with the 'fine ether tremors' that are a familiar phenomenon in light ether anesthesia, nor with the jactitations that may occur under ether when there is improper limitation of oxygen."

In his published request Blomfield[2] wondered if anesthetists in the United States had observed similar convulsive attacks during ether anesthesia, which, he said, seemed not to have occurred in England until recent times (i.e., in 1932) and for which, he said, no satisfactory explanation had been forthcoming. He reported that repeated clinical examination of the ether used yielded negative results, and he commented that convulsions form such a puzzling as well as a dangerous phenomenon that his committee was anxious to collect all information that might lead to an explanation for their occurrence.

The following year (1933) Kemp[3] of Canada pointed out the lack of a satisfactory explanation for the vigorous clonic muscular contractions that sometimes occur during ether anesthesia and that start with twitchings about the face and often extend to the muscles of the arms and even to those of the abdomen. He cited a case in which, after forty minutes of intratracheal ether-oxygen anesthesia, twitching of the face developed during closure of the abdomen. This seizure he assumed to be due to hyperventilation. He therefore practiced rebreathing to collect the patient's own carbon dioxide and administered oxygen through the anesthesia machine. These procedures checked the twitching of the facial muscles. When the same patient was operated upon again at a later date, twitching of the facial muscles developed fifteen minutes after anesthesia was started; these twitchings were again controlled by rebreathing (collecting the patient's own carbon dioxide) and by covering the face with an ether mask and damp cloths to effect a semi-open system. When the rebreathing was discontinued, twitchings recurred. Kemp postulated that the clonic muscular movements, sometimes seen in nonepileptic individuals under open intratracheal anesthesia, are due to general alkalosis, induced by the elimination of carbon dioxide with a consequent severe upset of the normal respiratory quotient. Noting that, if tetany developed in a patient during operation under ether anesthesia, tetany would develop in that patient if operated upon again at a later date, he concluded that the onset of tetany under ether anesthesia is due to factors inherent in the patient himself.

In 1936 Raab[4] of New York described convulsions occurring during ether anesthesia. That same year Payne[5] of London commented on the lack of medical literature on the subject of generalized convulsions that may occur in the course of ether anesthesia; he considered this phenomenon of serious importance, since it had occurred in no less than 200 cases during a ten year period, 1926 to 1936, and for which he stated a mortality rate of 22.2 per cent.

In 1937 Lundy[6] of Rochester, Minnesota, stressed the importance

2. Blomfield, J.: loc. cit.
3. Kemp, W. N.: Tetany during ether anesthesia. Anesth. & Analg. 12:1, Jan-Feb. 1933.
4. Raab, Abraham: Convulsions during ether anesthesia. Anesth. & Analg. 15:295-297, Nov.-Dec. 1936.
5. Payne, R. V.: Ether convulsions. M. J. Australia **24**:100, Jan. 16, 1937.
6. Lundy, J. S., and Tuohy, E. B.: General anesthesia complicated by convulsions. Surgery 1:666-687, May 1937.

of this problem of convulsions or spasms associated with anesthesia, which, he stated, were occurring with increasing frequency. While citing some thirty-three possible causes or significant factors involved in convulsions, he pointed out that, whether the etiology of a condition is known or not, the important thing is to know how to combat it when it arises. He suggested more careful choice of preliminary medication and of the anesthetic agent. He also proposed the intravenous injection of a soluble barbiturate for symptomatic treatment to control the convulsions and to reduce the fatalities, which, he stated, were more common than had been realized. He gave a mortality rate of 18.9 per cent at that time (1937).

In the Department of Anesthesia at Barnes Hospital during an eighteen year period, 1930 through 1948, convulsive seizures developed in fifteen patients during anesthesia. The details of some of these cases may be interesting as a background for the corrective procedures that I will outline later in this article.

CASE 1.—In 1930, during the course of an appendectomy being performed with the patient under nitrous oxide-oxygen-ether anesthesia, administered by the semi-open (valvular mask) method, progressive convulsive seizures developed during closure of the peritoneum. These started on the left side of the mouth and progressed to the left eyelid, left arm, and left leg. They then became generalized and violent; they were clonic in character and lasted for several seconds. The patient's temperature increased to 104 F. Fluids were administered, alcohol sponge baths were given, and oxygen was administered by the mask method. The seizures lasted for twenty-four hours, after which there were no recurrences. The patient was discharged from the hospital in two weeks.

CASE 2.—The following year (1931), during the course of a hysterectomy and associated bilateral salpingo-oophorectory being performed on a woman, aged 40, the second case of convulsions occurred. Two hours after the anesthesia had been started—nitrous oxide-oxygen-ether, administered by semi-open (valvular mask) method—facial twitchings developed in the same sequence that had been noted in the patient of the year before. The administration of the anesthetic was immediately discontinued, and oxygen was administered with 5 per cent carbon dioxide at intervals. Caffeine sodium benzoate, glucose, and blood transfusion were given, but the convulsive seizures continued. The patient died one hour and ten minutes after administration of the anesthetic had been discontinued, or three hours and ten minutes after the anesthesia had been started.

CASE 3.—In 1933 the third case of convulsions occurred in an adult male patient who had been admitted to the hospital with a gunshot wound. An abdominal exploration was being performed under spinal anesthesia when rhythmic jerking of the head developed. Inhalation anesthesia with nitrous oxide, oxygen, and ether was administered for twenty minutes until completion of the operation. The muscular twitching progressed to involve the entire body until the patient's death one hour and forty minutes after the spinal anesthetic had been given and fifty-five minutes after inhalation anesthesia had been started.

CASE 4.—During the year 1937 we had the fourth patient in whom convulsions developed during anesthesia. The patient, aged 27, was undergoing an appendectomy with drainage; the anesthetic agents were cyclopropane-oxygen-ether, given by the carbon dioxide-absorption method. After anesthesia had been under way one hour (forty minutes after the incision was made), convulsive seizures developed and followed the same course noted in the previous cases. Temperature increased to 41.8 C (axillary). Ice packs were applied, and calcium gluconate, saline solution, luminal, and oxygen were administered. The patient died two hours and twenty-five minutes after the induction of anesthesia was started.

With these unhappy experiences before us, we realized the desperate necessity for discovering some method of preventing the development of convulsions. The literature did not contain adequate information. We had tried the procedures that had been proposed but had

found them ineffective in the cases at hand.

The first important step forward revolved around recognition of the fact that the convulsive seizures generally developed during closure of the peritoneum and were presumably associated with reflex stimuli consequent to that procedure. We also noted that during, or as a result of, the convulsive seizure, the temperature increased in every case, in one case reaching 104.5 F. In view of this association of temperature increase with the onset of symptoms in each case, it was postulated that this type of convulsion was perhaps related to a corollary identifying disturbance of the heat-regulating mechanism. By then we had identified symptoms that we thought were characteristic and that would give the alert anesthetist adequate warning of the advent of an attack. With such warning the anesthetist could be prepared to prevent its development if corrective procedures were devised and if methods of applying them were at hand. The remedial procedures developed at that time (1937), and used by us since then, have proved quite effective both as preventive and as corrective measures. Since adopting them, we have not had a single fatality from convulsions. These procedures are as follows:

The preanesthesia temperature is carefully noted. If pyrexia, even of ½ degree, is present, an ice bag is placed under the nape of the patient's neck immediately after he loses consciousness, and administration of fluids is started at once. These precautionary measures usually prevent a further increase in temperature. Oxygen want is strictly avoided (as it should be under any circumstance), and it is verified that the carbon dioxide-absorption power of the soda lime is adequate.

Even if no pyrexia is present before anesthesia, the anesthetist is continually on the alert during anesthesia for the characteristic symptoms of preconvulsive seizures. At frequent intervals, about every ten minutes, the temperature is taken; with the development of the slightest elevation of temperature, the previously described measures are carried out. Since the described procedure has been put into routine effect and rigidly observed at the Barnes Hospital, we have not had a patient in our clinic in whom the violent convulsive seizures, described in the literature as "ether convulsions," have developed during anesthesia.

If the warning twitching around the mouth and eye (left side) does develop, a 2½ per cent solution of pentothal sodium is administered intravenously immediately, at a rate not faster than 1 cc. per minute. The twitching is usually under control after 2 or 3 cc. has been administered; rarely is it necessary to give more than 5 cc. of a 2½ per cent solution, the dosage range being 5/100 to 12/100 Gm. I repeat, however, that the alert anesthetist will not permit the condition to develop to the point that the convulsive seizure occurs.

The following symptoms are characteristic of approaching convulsive seizures during anesthesia: an elevation of temperature of from ½ to 1 degree, associated with an elevated blood pressure; and tugging or pulling of the muscles of the throat, which can be readily detected by the hand of the anesthetist in supporting the jaw. Cyanosis is not present, nor have we observed any characteristic change in the rate or depth of respiration, nor any appreciable change in the rate or volume of pulse.

These symptoms of approaching

convulsions develop from one half to one hour before convulsions develop. If these early warning symptoms are not recognized, there will be a gradually progressive development of a more ominous nature. The temperature will progressively increase to 102-104 F.; the tugging or pulling of the throat muscles will become more pronounced. It is important to remember that, if this latter symptom were considered only by itself, it could be mistaken for a reaction to an overdose of the anesthetic (ether). On the basis of such a mistaken interpretation of the cause, the ether tension would be lowered and additional oxygen administered; these procedures, while indicated for overdosage of the anesthetic, would not correct the condition at hand if it were in reality a warning of an approaching convulsive seizure and not a symptom of overdosage. In the case of such misinterpretation of the cause and consequent attempts to correct it, further symptoms would develop, namely, twitching of the muscles of the left side of the mouth, progressing to the left eye, the left extremities, and on to generalized convulsive seizures of great intensity, which would interfere with the operative procedure and even possibly terminate fatally.

It is also very important that the symptoms warning of the approach of the convulsive seizures referred to here (those possibly related to a disturbance of the heat-regulating mechanism) should not be confused with the symptoms that warn of approaching convulsive seizures due to oxygen want. These latter are: (1) rapid, shallow respiration, becoming jerky and irregular, followed by gasping, slow respiration; (2) general tonic muscular rigidity, jactitations, and opisthotonus; (3) elevated blood pressure with rapid, weak pulse; (4) markedly dilated pupils without reaction to stimulation by light; (5) cyanosis with arterial blood of venous color.

If such an inexcusable condition should be permitted to develop, it can be corrected by the administration of oxygen and manual compression of the breathing bag and the maintenance of free respiratory exchange.

COCAINE POISONING

Let us now turn to a different type of complication that not infrequently faces the anesthetist for correction, although she is usually not present when the unfavorable reaction begins. I refer to those eventualities that develop following the use of cocaine or of one of its derivatives, such as pontocaine, procaine, nupercaine, and butyn. The use of cocaine or its derivatives is advocated by some authorities to prevent reflex closure of the glottis during the introduction of an intratracheal catheter. Some of the champions of the drug even narcotize the larynx and throat with cocaine to the extent that an intratracheal catheter may be introduced without administering any inhalation anesthetic whatever.

Procaine or other cocaine derivatives are used by some anesthetists as a throat spray in preparation for a bronchogram or bronchoscopy. The drug also is used locally in nasal surgery. The injection of procaine into the heart has been advocated for cardiac arrest, as has the intravenous use of procaine for the prevention and treatment of cardiac arrhythmias during anesthesia and as a protection against ventricular fibrillation. Reports concerning these intravenous uses of procaine are very conflicting.

Cardiac arrhythmias and cardiac arrest, occuring during thoracic operations, remain a dreaded and occasionally fatal catastrophe, and many clinical observations have been made during these episodes. However, despite the considerable amount of investigative work done on the problem, there is even now no unanimity of opinion as to the cause or the treatment. According to one group, manipulation of the lung root or any segment of the heart or great vessel is likely to set up vago-vagal reflexes that touch off an arrhythmia or cause cessation of the heartbeat. It is this group that believes that great benefits derive from the use of procaine intravenously, as well as from the topical use of cocaine or pontocaine, both as a prophylactic and as a curative measure.

At Barnes Hospital there is considerable skepticism in regard to the practical importance of vago-vagal reflexes. Graham,[7] on the basis of an extensive experience in thoracic surgery and dog experiments, doubts the existence of true vago-vagal stimuli as a *modus operandi* of cardiac arrhythmias and arrest. Burford[8] believes that myocardial ischemia is a more likely mechanism and doubts that arrest or serious irregularities occur in hearts previously undamaged by disease. In the few cases of arrhythmia and/or arrest that have occurred on the Chest Service at the Barnes Hospital, Graham and Burford have contended that cardiac massage is the one dependable method for restoring the beats; and that, if this measure fails, no adjunctal expedient, including electrical stimulation, is likely to be of benefit. They place small reliance on the administration of pontocaine locally or of procaine intravenously. Instead, they stress smooth anesthesia with maximal oxygenation and gentle operative technics.

While there is no question that cocainization prior to intratracheal intubation facilitates the intubation procedure, the practice is considered by some to be accompanied by considerable danger. It is therefore strongly urged that, instead of using cocainization as a preparation for intubation, a general anesthetic be administered to secure a plane of anesthesia that will abolish the afferent impulses, which under too light a plane of anesthesia are active and interfere with introduction of the intratracheal catheter.

In our institution the spraying of the throat of a patient scheduled for a bronchogram or bronchoscopy was from time to time attended by a high incidence of convulsions and other symptoms before we adopted the practice of routinely administering nembutal prior to the cocainization. However, even after these precautionary measures had been inaugurated, untoward symptoms developed all too frequently.

Many years ago, two deaths occurred in our clinic in patients cocainized for nasal surgery, and one death occurred in a patient prepared for a bronchogram. In all three cases, the patients suddenly stopped breathing, were pulseless upon the arrival of the anesthetist, and failed to respond to resuscitative measures. A recent fatality occurred in a patient who had been prepared by local infiltration block for an abdominal operation. Immediately after the injection of 1 per cent novocain, the patient's head began to jerk, and a generalized convulsive seizure developed of such great violence that pentothal

7. Graham, E. A.: Personal communication to the author.
8. Burford, T. H.: Personal communication to the author.

sodium could not be administered intravenously, and, although an intratracheal catheter was introduced and oxygen given through it by manual compression of the breathing bag, pulse and blood pressure became imperceptible, and the patient was pronounced dead twenty minutes after the novocain injection. Several cases of convulsions occurred in patients whose throats had been sprayed with procaine prior to bronchoscopy; typical symptoms of cocaine poisoning developed, namely, shortness of breath, resistance, restlessness, fast pulse, rigidity, anxiety, muscular twitchings, elevated blood pressure, and pallor, followed by cyanosis. In each of these cases, however, the symptoms were controlled by administration of pentothal sodium plus oxygen, and in each case bronchoscopy was performed without further incident. All of these patients had previously been prepared with 0.09 Gm. of nembutal administered an hour before bronchoscopy.. Waters and Gillespie[9] reported "several deaths after topical application of a drug to the pharynx," but as no member of the department of anesthesia was present when the deaths occurred, no records were available.

Since the anesthetist is called upon to assist in emergencies that arise after cocaine administration, she should familiarize herself with the symptoms of cocaine poisoning and the method of treating them, as well as with precautionary measures that may be inaugurated prior to cocainization. The symptoms of cocaine poisoning and poisoning from its substitutes are similar: dizziness, marked pallor, dilated pupils, nausea, vomiting and abdominal pain, finally delirium, dyspnea, slow weak pulse, and convulsions. Fatal cases run a very rapid course, with anxiety, sudden fainting, extreme pallor, dyspnea, sometimes brief convulsions, arrest of respiration, and death. The prior administration of a barbiturate diminishes the risk by minimizing the cocaine convulsions and their effects when they develop.

In our clinic 0.09 Gm. pentobarbital sodium (nembutal) is administered two hours before the operative procedure. Alternative recommendations that have been made are: 0.2 Gm. phenobarbital or 0.6 Gm. sodium barbital one hour before operation.

If symptoms of cocaine poisoning develop, the patient's head is lowered, and the convulsions are arrested by the intravenous injection of pentothal sodium. Artificial respiration is administered (usually an intratracheal catheter is introduced), and cardiac massage is instituted if the circulation fails.

COMPLICATIONS DURING THORACIC OPERATIONS

I shall refer briefly now to procedures associated with the conduct of thoracic operations. It is axiomatic that, first, adequate pulmonary ventilation must unfailingly be maintained at all times and, second, that the possibility is always present that, as a result of a manipulative procedure, lung exudate may be released into the tracheobronchial tree.

Accordingly, at Barnes Hospital, where there is a major clinic in thoracic surgery, anesthesia procedures have been set up, the details of which may be interesting.

Immediately after introduction of the intratracheal catheter, suction is effected through it. This suction is again thoroughly effected

9. Waters, R. M., and Gillespie, N. A.: Deaths in the operating room. Anesthesiology 5:113-128, March 1944.

just prior to turning the patient from the supine to the lateral position. Suction is again promptly effected through the catheter immediately after the patient is turned, if indicated.

It is to be remembered that, after the incision has been made and during rib resection, there is likely to be a release of purulent material into the bronchial tree. Accordingly, it is a routine procedure with us to use suction at that time, as well as during the opening of the pleura. Again, during actual manipulation of the lung, there are periods when excessive material may be forced into the respiratory tract. At such times the tracheal catheter is routinely suctioned.

Throughout the operation, the anesthetist is on the alert for moist sounds; should they develop, she does not hesitate to ask the surgeon to interrupt the operation to permit needed suction. In cases of suppuration it is especially necessary that suctioning be done at frequent intervals. While these suctionings may interfere with and delay the operative procedure, it must be realized that any secretion permitted to be present in the air passages will interfere with the gaseous exchange in the lung and, if not removed, may lead to severe anoxia, with its accompanying detrimental effect on cardiocirculatory functions.

Upon completion of the operation and after deanesthetization, the patient's mouth is thoroughly suctioned, and the suction catheter is introduced into the trachea to aspirate secretions from the lung, as well as normal secretions or mucus that may be present in the trachea. After this procedure the normal cough reflex is usually present and aids in the avoidance of postoperative pulmonary complications.

From the foregoing, it will be apparent that we consider it imperative for the anesthetist to have at hand at all times during anesthesia a properly functioning suction apparatus that may be instantly used if and when needed. I repeat for emphasis that, if secretory or mechanical obstruction is permitted to exist, various degrees of anoxia will supervene, with consequent depression of the central nervous system and resultant hypertension and tachycardia during anesthesia, as well as postanesthesia pulmonary complications, such as atelectasis and bronchopneumonia.

COMPLICATIONS CAUSED BY REFLEXES

I cannot leave the subject of complications of anesthesia without briefly referring to those that revolve around anoxia caused by laryngeal and other reflexes, which interfere with untrammelled respiratory exchange and which, if permitted to continue, eventuate in myocardial exhaustion, cerebral damage, and even death.

As Brewer and Luckhardt[10] pointed out, laryngeal reflexes are divided into two classes, first, those that originate in the larynx itself and, second, those that result from stimulation of afferent nerves elsewhere in the body. An illustration of this latter reflex action is the cessation of respiration—blocking off of respiration by approximation of the vocal cords—that results from traction on the splanchnic viscera during an abdominal operation, if the plane of anesthesia is permitted to become unnecessarily light for the surgical procedure be-

10. Brewer, Nathan.; Luckhardt, A. B.; Lees, W. M., and Bryant, D. S.: Reflex closure of the glottis by stimulation of afferent (visceral) nerves. Anesth. & Analg. 13:257-259, Nov.-Dec. 1934.

ing performed. The vital importance of maintaining anesthesia at sufficient depth to avert the supervention of such traction-stimulated, reflex closure of the glottis must be recognized, and such complication inviolably avoided. Another illustration of the occurrence of laryngeal reflex because of too light anesthesia is the severe laryngeal spasm, with complete adduction of the vocal cords, that occurs if surgical intervention is undertaken too early, i.e., before the patient has fully reached the plane of anesthesia necessary for the procedure at hand.

Obviously, different surgical procedures call for different planes of anesthesia for their safe execution. Different stages of a given operation also require different planes of anesthesia. In my experience, one of the best ways of verifying that the plane of anesthesia is proper for the procedure at hand is the change in character of the respiration, which clinically manifests the various zones during anesthesia. During the induction period respirations do not vary greatly from normal, but their change in character with loss of consciousness and supervention of the second stage must be studied critically if the characteristic increase in rate and in depth that is typical of entry to that stage is to be identified. This change is easily noted when the anesthetic agent is ether, but it is less noticeable when the anesthetic agent is one of the quicker acting gaseous agents, nitrous oxide-oxygen or ethylene-oxygen. When the anesthetic is cyclopropane-oxygen, this increase in rate and depth of respiration is neither marked nor of long duration, so that the anesthetist must be especially alert to identify and interpret its occurrence. After the increase in rate and depth of respiration that occurs at this point, a characteristic type of respiration develops, which, for lack of any formal description, I describe for my anesthesia students as "superficial and hesitant in character, with a slightly shorter inspiratory phase and a slightly longer expiratory phase and with a slight pause between those inspiratory and expiratory phases."

The anesthetist must not misinterpret this characteristic "superficial hesitant" respiration as the "unequal inspiratory and expiratory phases" of respiration that indicate approaching overdosage; such misinterpretation would result in the lightening of the plane at this point with activation of the vomiting reflex to produce vomiting, with the danger of aspiration of the stomach contents into the trachea, possibly leading to dire consequences. Nor should surgical intervention be undertaken at this stage of anesthesia, because of the reflex closure of the glottis and laryngeal spasm that would result.

After successful transition through the "superficial hesitant" respiratory phase of anesthesia that I have just described, a type of respiration delevops that is indicative of the establishment of the surgical plane of anesthesia. This is characteristically automatic, rhythmic, and machine-like, with the inspiratory and expiratory phase of nearly equal length and with no noticeable pause between these two phases.

A pharyngeal airway and intratracheal catheter of appropriate size should always be at hand and used if needed to insure mechanically unobstructed respiratory exchange. An anesthetist's boast of seldom using an airway is a confession that that anesthetist is not alert

to the importance of this principle of anesthesiology and of the damage that can result from violating it.

Complications During Upper Abdominal Operations

In concluding this article, I shall make some recommendations concerning the avoidance of some crises that are reported to arise during anesthesia for upper abdominal and thoracic surgery. At Barnes Hospital routine procedures for caring for patients undergoing these types of operation have reduced to a minimum certain complications that we know from the literature otherwise occur frequently.

The surgical procedures to which I refer are gastric and intestinal resections and operation for duodenal perforation, intestinal obstruction, etc. One of the untoward circumstances I have in mind is the ejection of the stomach contents into the mouth and respiratory tract during anesthesia. I have also in mind the release of lung exudate into the tracheobronchial tree when manipulative procedures are being performed during thoracic operations; the presence of mucus in the upper respiratory tract as the result of the secretion of saliva during early stages of anesthesia; and the presence of a plug of mucus in the bronchus following anesthesia.

To reduce to a minimum the occurrence of such complications, the Barnes Hospital Department of Anesthesia has set up certain technics that have proved quite effective.

Every patient scheduled for a gastrointestinal operation has a duodenal tube introduced prior to the administration of the anesthetic. Usually this tube is in place when the patient arrives in the operating room. If for any reason this procedure has *not* been executed, the anesthetist requests that she be permitted to introduce such a duodenal tube. Generally the surgeon is most appreciative of the suggestion.

Then, prior to the administration of the anesthetic and while the patient is still conscious, the duodenal tube is suctioned thoroughly. Experience has shown that, even though a duodenal tube is in place and draining, as much as 1,000 cc. of fluid, and usually 500-700 cc., may be obtained by suction. After suctioning, the open end of the duodenal tube is placed in an emesis basin, which has previously been strapped securely to the operating table by means of adhesive, so that the pan will not fall to the floor at an inopportune time during the operation.

The patient is then given pentothal sodium intravenously until he loses consciousness, after which, an inhalation anesthetic is administered. As soon as the eyelid sign is abolished, an intratracheal catheter is introduced orally under direct vision. The balloon on the catheter is inflated to the proper tension, causing the sides of the balloon to press gently against the walls of the trachea, thereby preventing any fluids in the mouth from invading the respiratory tract. The anesthetic of choice is then administered by the carbon dioxide filtration, closed circuit technic.

The duodenal tube is frequently suctioned during the operative procedure, especially if there is considerable fluid flowing from the tube. This is important, because during manipulation of the digestive tract, the amount of fluid forced out through the esophagus is sometimes greater than can be carried away by the duodenal tube. By fre-

quent suctioning, however, the amount of fluid in the digestive tract is kept at a minimum, thus reducing the possibility of digestive fluids' being suddenly forced into the mouth.

While a patent airway is assured by the presence of the intratracheal catheter, the mouth must be kept free of any digestive fluid in preparation for the time when the catheter will be removed and when some fluid in the mouth might reach the respiratory tract in spite of the fact that thorough suctioning has been done.

While we have for many years used the intratracheal technic of anesthesia for gastrointestinal surgery, only during the past few years have we taken the detailed precautionary measures described. I cannot stress too emphatically the importance of following these comparatively simple procedures to prevent possible aspiration into the trachea of stomach contents that may be forced into the mouth during surgical manipulation.

SUMMARY

By the adoption and meticulous execution of protective technics such as I have outlined in this article, together with appropriate postoperative care, we have reduced postoperative pulmonary complications to an incidence of 0.0028 per cent (4 cases out of a total of 14,157 during the year 1948).

I therefore propose that the procedures that I have set forth in this article be combined with those already in effect in departments of anesthesia of progressive institutions, to reduce to a minimum the incidence of certain complications during anesthesia and to make smoother and less eventful the course of anesthesia generally.

ANOXIA

(*Continued from page* 86)

garding the nature of cerebral changes occurring after acute periods of anoxia in human beings. *Permanent damage to the brain as a result of acute cerebral anoxia not only is a function of the duration of the anoxic episode, but also, in the case of sublethal periods of anoxia, must be conditioned by the duration and severity of a period of postanoxic cerebral edema.* It is suggested that a considerable portion of the permanent cerebral damage reported in these unfortunate cases may be the result not of the initial insult, but rather of a period of untreated cerebral edema subsequent to the initial insult. If this hypothesis is accepted then it must follow that treatment should be directed toward the alleviation of the cerebral edema secondary to acute anoxic accidents. We suggest that the most effective treatment of this kind presently available is the intravenous administration of comparatively large doses of 25 per cent human serum albumin at frequent intervals.

At present animal studies are in progress to determine whether a sound, experimental basis can be established for the hypothesis proposed. If these observations and comments are valid, then the advisability of similar treatment in such cases as acute poisoning with barbiturates and other depressant agents must be considered as adjuncts [sic] to other therapeutic measures.

SUMMARY

An attempt has been made to review the literature pertaining to anoxia and its effect on the cerebral cortex.* In this survey it was not intended to draw any positive conclusions. The opinions of the authors as to the importance of anoxia varies. That the subject has a continuing interest is indicated by current reports of investigation and experiment. The whole answer may never be known, but the mass of evidence that is being accumulated should eventually lead to the clarification and elimination of these devastating episodes.

*Parts I and II of this review were published in the May and August issues of the JOURNAL in 1949.

CHILDREN'S FEAR AND ANESTHESIA
With Emphasis on "White Syncope"

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Children are afraid of inhalation anesthesia for many reasons. They fear it for the same reasons many adults do, namely, the possibility of death or suffocation.[1] They also fear it because they do not understand it: They do not understand how they can be cut or undergo other equally painful ordeals and yet not wake up. They do not understand why they will not be able to wake up whenever they want to, nor what—if they are a little older—it is that keeps them from waking up during the operation. These fears may be voiced, but, according to Jersild,[2] a child, as he grows older, conceals the visible expressions of his fears. Also, a child may fear strangers. Even an infant 5 or 6 months of age may show fear of strangers. A visit to the doctor, the first visit from a grandmother or an uncle, or even the appearance of a new babysitter will cause many an infant of this age to cry lustily, especially if the new acquaintance attempts to handle him. A visit to the hospital, where everyone is strange, may cause great fear and anxiety in children from this age group up until preadolescence. If the anesthetist has not seen the child prior to the induction of anesthesia, she may expect the child to fear her also. Jersild[2] also stated that "with the development of the child's imaginative abilities, his fears become increasingly concerned with imaginary dangers." Many children are told that deceased persons are "just asleep," and the thought of enforced sleep from anesthesia is promptly related, and the fear of death evolves. Other children may tell the patient distorted stories of operations. In such a case, the child is usually apprehensive because he knows that he will not be able to protect himself after he is asleep. An apparently composed boy of 11 years once asked me, quietly and seriously, through the fog of barbiturate sedation, "Is the doctor really going to cut my leg off, too?" It was his first operation, and he was to have his tonsils removed. The other boys on his ward told me later, "Oh, we didn't think he'd believe us; he didn't seem to."

Although a child's fear of anesthesia makes him unhappy, it is the physiologic reaction to fear that may be detrimental. According to Salder,[3] "The ductless glands are undoubtedly affected by the states of both acute fear and chronic worry." Jersild[2] stated that a hypodermic injection of adrenalin will produce the same bodily changes as are seen in an individual suffering from fear. Guedel[4] was more spe-

1. Dunbar, Helen F.: *Emotions and Bodily Changes* (New York: Columbia University Press, 1946).
2. Jersild, A. T.: *Child Psychology* (New York: Prentice-Hall, Inc., 1946).
3. Salder, W. S.: *Theory and Practice of Psychiatry* (St. Louis: C. V. Mosby Company, 1936).
4. Guedel, A. E.: *Inhalation Anesthesia* (New York: The Macmillan Company, 1938).

cific and stated that:

Emotional excitement—fear—produces an increased activity of the sympathetic nervous system and consequently an increased output of adrenin from the suprarenals. This sets the stage for fibrillation in response to any added stimulus such as the delirium of the second state of anesthesia, voices or any physical disturbance of the patient during the induction period.

Dunbar[1] observed that many apparently normal, healthy persons died on the operating table "even before the anesthetic was begun," owing to emotionally instigated factors. Furthermore, *The Cyclopedia of Medicine*[5] states that the beginning of paroxysmal auricular fibrillation may be due to nervous hypersensitiveness, which predisposes to the arrhythmias in an apparently normal person, and that it "is important that the child be free from alarm or fright." Dogliotti[6] found that the more anxious and frightened the patient was, the more likely cardiac syncope was to occur. Also he did not think that the anesthetic causes cardiac syncope, because it usually occurs during induction when there is a low concentration of anesthetic circulating in the blood stream, but that it is probably due to a "nervous mechanism" such as "a reflex inhibition of the vagi which caused a rapid and pronounced arrest of cardiac function." *The Cyclopedia of Medicine*[5] states that adrenalin, by increasing intracranial pressure, may stimulate the vagus, which is partly responsible for the arrhythmias and extrasystoles that follow its injection; by direct action, it increases the irritability of the ventricular muscle fibers; and by stimulating the sympathetic nervous system, it speeds up and increases the contractions of the heart by activating the pacemaker and increasing the speed of conduction of stimuli. "In fact, acute fatal cardiac failure from auricular fibrillation may result in this way, especially if adrenaline is administered during light chloroform narcosis."

The necropsy report of "status lymphaticus" may unintentionally cover up the true diagnosis of fatal auricular fibrillation. It is no doubt a welcome crutch to the anesthetist who has a sudden dramatic death on the operating table in an apparently healthy, normal child undergoing an elective operation. Although "status lymphaticus" has been listed in many postmortem reports as the cause of sudden death during induction of anesthesia, authorities are more or less equally divided on the probability of it as an entity.[4] Pathologically, "status lymphaticus" is described as a large thymus and congested adrenal glands. Leigh and Belton[7] reported one anesthetic fatality due to respiratory obstruction from a known cause in which this same description of pathology was given. Adriani[8] made the statement that "No relationship has been established between any secretion of the thymus gland and the so-called *'status lymphaticus'* and anesthesia." Dogliotti[6] allowed for alternatives, when referring to the "characteristic white syncope" that precedes death early in inhalation anesthesia, by stating that it is due to "status thymus lymphaticus," to a marked congenital hypoplasia of the cardiovascular system, or to lesions found in the adrenal glands.

1. Dunbar, Helen F.: loc. cit.
5. *The Cyclopedia of Medicine*, ed. 1, 1936, I, 189 and 456.
6. Dogliotti, A. M.: *Anesthesia* (Chicago: S. B. Debour, 1939).
4. Guedel, A. E.: loc. cit.
7. Leigh, M. Digby, and Belton, M. Kathleen: *Pediatric Anesthesia* (New York: The Macmillan Company, 1948).
8. Adriani, John: *The Chemistry of Anesthesia* (Springfield, Ill.: Charles C Thomas, 1946).

Nosworthy's[9] description of some cases of "primary cardiac failure" that occurred suddenly during induction — i.e., "The complexion turns ashy pale, the pulse is imperceptible, the pupils are widely dilated and inactive, and the breathing ceases after a few shallow breaths"—is also a good description of Dogliotti's[6] "characteristic white syncope" and "status lymphaticus."

If "status lymphaticus" is not an entity, there are even more sudden deaths during induction of anesthesia to explain without help from the necropsy report. In deaths from auricular ventricular fibrillation the postmortem examination shows only one pathologic finding: "Death from ventricular fibrillation is due to cardiac anoxia." The heart will stop in full diastole according to Guedel,[4] who described it as:

> Cardiac ventricular fibrillation is a worm-like movement of the ventricular muscle with the heart in full diastole. It consists of an unsynchronized contraction and relaxation of the individual fibers, which provides no circulation of blood. Its occurrence depends upon an increased activity of the sympathetic nervous system and the resultant increase of adrenin in the blood, together with a probable increase in the irritability of the ventricular muscle itself. The adrenin may be poured into the blood stream by the suprarenal glands or it may be introduced from without.

Starling[10] and Adriani[8] agree that adrenalin is secreted during (and because of) general anesthesia, and many authorities agree that fear or nervousness increases the action of the suprarenal glands. As previously mentioned, adrenalin increases the irritability of the ventricular muscle fibers directly, and, reflexly, it may be partly responsible for the arrhythmias and extrasystoles that follow its injection.[5] If more adrenalin is added during the occurrence of these abnormal ventricular rhythms of the prefibrillation type, fatal ventricular fibrillation may occur.[11] Adriani[8] also stated that adrenalin stimulates the sympathetic system, which accelerates the heart action through the pacemaker.[11] A frightened child will attempt to hold his breath during induction, and the resultant oxygen want will increase cardiac irritability.[12] Thus many authorities agree that stimulating the pacemaker, when the irritability of the heart muscle has been increased, makes auricular ventricular fibrillation possible. Since adrenalin may cause both these conditions and since three separate factors — fear before anesthesia, general anesthesia, and a prolonged excitement stage — may stimulate the flow of a great deal of adrenalin, it is logical to assume that here is the answer to "characteristic white syncope" that occurs so dramatically during induction of anesthesia but produces no definite, pathologic condition to be discovered on postmortem examination.

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9. Nosworthy, M. D.: *The Theory and Practice of Anesthesia* (London: Hutchison Scientific, 1935).

6. Doglotti, A. M.: loc. cit.

4. Guedel, A. E.: Loc. cit.

10. Starling, E. H.: *Principles of Human Physiology* (Philadelphia: Lea & Febiger, 1933).

8. Adriani, J.: loc. cit.

5. Loc. cit.

11. Goodman, Louis, and Gilman, A.: *The Pharmacological Basis of Therapeutics* (New York: The Macmillan Company, 1941).

12. Adriani, John: *The Pharmacology of Anesthesia* (Springfield, Ill.: Charles C Thomas, 1942).

COMPLEX MEDICAL PROBLEMS IN RELATION TO ANESTHESIA

The forum on "Complex Medical Problems in Relation to Anesthesia," at the Sixteenth Annual Meeting of the American Association of Nurse Anesthetists, was called to order in the Music Hall, Public Auditorium, Cleveland, Wednesday afternoon, September 28, 1949, at 2:15 p.m., Miss Lou Adams, chairman, presiding.

CHAIRMAN ADAMS: The meeting this afternoon is to be devoted to a forum on "Complex Medical Problems in Relation to Anesthesia." We have had some questions sent in by mail. We will take them up first. We have provided you with papers. Please write your questions and pass them to the aisle, and the ushers will pick them up.

It is up to you to make this meeting a success and a most interesting one. I have known this outstanding group of doctors for many years, and all are from the Cleveland Clinic Hospital.

It is my pleasure to introduce to you Dr. A. Carlton Ernstene, Director of the Medical Department; Dr. E. Perry McCullagh, Metabolic Diseases; Dr. Donald B. Effler, Thoracic Surgery; Dr. Robert D. Taylor, Research; Dr. Donald E. Hale, Director of the Department of Anesthesiology. Dr. Hale will now act as moderator.

DR. HALE: Members of the American Association of Nurse Anesthetists and guests: We hope you will follow Miss Adams' suggestion and really make this program a success by asking questions and asking them promptly. Instead of calling for questions from the floor, we ask you to write them out so they may be read here and heard readily by everyone. It should be pointed out that at the end of the meeting we shall be gone, and the opportunity will go with us. Don't expect to waylay us on our exit and get in your final questions. Please get them on paper and to the aisles where they will be collected at intervals during the program.

While you are formulating your questions and writing them down, we will start by taking a few questions that have already been submitted.

What is the best management for patients with heart disease requiring emergency surgery?

I will ask Dr. Ernstene to discuss this problem.

DR. ERNSTENE: Dr. Hale, I think there still is a rather widespread misapprehension about the strain that anesthesia and an operation place upon the heart. Actually, the heart is called upon to perform no appreciably increased work during an operation and under anesthesia. The two great dangers, of course, to which the patient is subject are, first of all, danger of anoxia and, second, danger of hemorrhage. If these are avoided,

I believe that it may be accepted as a general rule that a patient with organic heart disease is able to tolerate an operation and anesthesia without undue risk, if he has been able, prior to operation, to carry on the activities of everyday life without shortness of breath and without pain in the chest.

If a patient has congestive heart failure, on the other hand, there should be, if possible, a period of preoperative preparation with digitalis and other measures to overcome the failure. If time will not permit adequate treatment and there is a true emergency, rapid digitalization is in order. A patient can be completely digitalized within a very short time, and if this is accomplished, most patients will go through an operation under anesthesia without difficulty.

There are a few types of heart disease that are attended by the risk of sudden death. These conditions are relatively uncommon and include complete or high grade heart block, certain diseases of the aorta with involvement of the aortic valve, angina pectoris, and recent acute myocardial infarction. In these particular cases great care must be exercised to avoid anoxia during operation, and even then, despite all measures, serious complications or even sudden death may occur.

However, I think that if a patient is able to carry on ordinary daily activities without difficulty, even though physical examination may show evidence of definite organic heart disease, he may be expected to undergo anesthesia and a surgical operation without difficulty.

Dr. Hale: Thank you, Dr. Ernstene. I should like to point out that, if these questions I am reading now or subsequent ones or any of the answers bring to mind fresh questions, we shall be glad to have them submitted also.

Here is a question for Dr. McCullagh.

Should glucose solution be given to a diabetic patient?

Dr. McCullagh: Mr. Chairman, the problem of giving glucose solution to a diabetic patient revolves chiefly about two parts of the diabetic problem. One is whether the patient has gone a long time without food, particularly without carbohydrates, and, secondly, whether he has had insulin that is still active at the time under consideration.

If the diabetic patient is operated on under normal circumstances, all preparations having been made routinely, there is no particular advantage in giving glucose solution during the operation.

If the patient has had his morning dose of insulin—whether it be crystalline zinc insulin, protamine insulin, or globin insulin—and for some reason requires an operation unexpectedly, because of an accident or a similar emergency, and if he has not had food that would ordinarily be covered by the insulin, or if he has vomited, then one must consider providing the needed carbohydrate as glucose. Under these circumstances glucose should certainly be given during an operation.

If glucose solution is given under such circumstances, it is better to give more than sufficient glucose rather than less. There is no particular harm in increasing the blood sugar level to moderate heights during an

operation or immediately thereafter. We have no fear whatever of blood sugar levels of 200, 300, or even 400, although we prefer to keep them within an easily manageable range.

The main issue in considering high blood sugar levels is not the blood sugar itself when circumstances favor correction in a few hours or a day, but whether or not the patient is susceptible to ketosis or acidosis. In other words, is the diabetes under control, even though the blood sugar level may be temporarily increased, or has it been out of control?

There are several other more or less related problems on which I might comment. One is the problem of giving glucose solution during operation to a patient who has liver disease. It is sometimes difficult to determine whether a diabetic patient has liver disease, or whether the blood sugar levels are increased because the liver, diseased from other causes, fails to absorb the glucose from the blood, the glucose reaching high levels and the condition simulating other types of diabetes.

In general, however, the liver functions poorly on an inadequate intake of carbohydrates. The substance is depleted rapidly. Therefore, when there is definite or suspected liver disease, or when the liver is enlarged, or when there is biliary infection or a suspicion of liver disease in a patient requiring an operation on the gallbladder, there may be some advantage in giving carbohydrates, such as glucose, intravenously during the operation. The most important thing is to keep the carbohydrate content of the blood high, and if carbohydrates can be given before operation, in quantities of 200, 300, or even 400 Gm. each day, the question of giving glucose solution during operation is unimportant.

Adrenal failure is seldom encountered. A patient with Addison's disease rarely requires operation; unfortunately, when one does, the condition frequently has not been recognized previously. If a patient known to have adrenal failure of the Addison's disease type requires operation, one should remember that hypoglycemia under the circumstances is poorly tolerated. The adrenal hormones are important in giving the body the power to restore low blood sugar levels to normal and to create sugar for the blood from sources other than the carbohydrate stores of the liver. When the blood sugar level falls too low in a patient with Addison's disease, this hypoglycemia cannot be corrected, and the patient is likely to die suddenly as a result. Under the circumstances mentioned, carbohydrates should be given intravenously during operation and in some manner subsequently.

The same is true in instances of pituitary failure, in which the adrenal gland, by reflecting the deficiency of the pituitary gland, loses at least a portion of its power to restore blood sugar levels to normal. In some cases in which the pituitary gland is being operated on, serious hypoglycemia is not to be expected within a few hours. But in the case of chromophobe adenoma, suprasellar cyst, or perhaps adenoma in the region of the pituitary, a circumstance in which the active cells of the pituitary may be pressed upon to the point of loss of function, hypoglycemia is to be watched for and avoided. In discussing this type of hypoglycemia and its avoidance, it

is well to remember that when glucose solution is given to a patient with Addison's disease or with pituitary failure, blood sugar increases during the time of the intravenous administration. However, glucose also has the power of stimulating the output of insulin within the body, so that three or perhaps four hours after the administration of glucose is stopped, the blood sugar is likely to decrease to levels considerably lower than before the administration. In other words, one can, by giving glucose intravenously, introduce the factor of hypoglycemia later, which under these circumstances may be fatal. It must be clearly understood that if glucose is given intravenously during operation under these circumstances, glucose must also be given after operation, preferably by mouth, but otherwise by vein, to avoid later hypoglycemia.

DR. HALE: Thank you Dr. McCullagh. I have a question here for Dr. Taylor.

What is the probable cause of sudden respiratory and circulatory failure following reduction of a fractured femur? The tourniquet had just been removed, and until that time the condition of the patient was apparently good.

DR. TAYLOR: There are several things one would have to consider as being the cause of sudden collapse and circulatory and respiratory failure in a patient under these conditions. Among the ones that most quickly come to mind is fat embolism. However, emboli are not usually widespread enough or severe enough to cause complete failure. Rather, the course is slow deterioration of the nervous system after the occurrence of such emboli.

An air embolus might have occurred if it were an open reduction, as I presume it was. Under these conditions large venous channels are often widely open, and negative venous pressure sucks in air.

In all probability the condition is the same one sees during shock. During World War II we developed a method of producing shock in animals, which was easily reproducible. We placed tourniquets around both thighs for periods of one to four hours. When these tourniquets were released, there was a precipitous decrease in blood pressure, and fatal shock ensued. The mechanism is not completely explained even now. Several things happen. First, a large vascular bed in the legs is taken out of the circulation for a long period. On reopening the circulation of the extremity, the sudden outpouring of blood into the legs amounts to a large hemorrhage.

It has been shown in human beings that one can pool between 500 and 1,600 cc. blood in an extremity by obstructing the venous return. If one does this to a patient who has already been subjected to a traumatic lesion, such as a fractured femur, which in itself is capable of producing shock, we have a situation that produces profound shock and circulatory collapse.

Dr. Best of Canada, who is using this method of producing shock, cannulated the lymphatic vessels that drained areas subjected to long application of a tourniquet and found that the lymphatic fluid yielded a

substance that, injected into an apparently healthy animal, induced a rather profound decrease in blood pressure. In some opinions, a bacterial contaminant was believed responsible.

We have, therefore, a combination of fractured femur, blood loss, and wound; the tourniquet applied for a few minutes or an hour or two; and this unknown substance that is drained into the general circulation, all contributing to the production of surgical shock.

DR. HALE: Here is a question for Dr. Effler.

Please discuss the importance of positioning in thoracic surgery.

DR. EFFLER: I think positioning in thoracic surgery probably boils down to one important feature as far as the anesthetist is concerned, and that is the maintenance of an adequate airway.

As you know, most thoracic surgeons prefer to operate upon a patient with the involved, or diseased, side up and the patient lying on his other side. Others prefer to operate with the patient in the prone or the supine position. I think from the standpoint of anesthesia the important thing is to have the patient in a position that will satisfy the operating surgeon—this, I believe, is the first duty of any good anesthetist—and, secondly, to have him in a position in which an adequate airway can be maintained and the tracheobronchial tree aspirated freely.

When a patient has a suppurative disease, such as a tuberculous or abscess cavity, and the good side is down and the affected side up, as in the most conventional position, there is an excellent opportunity for secretions to drain into the underlying sound lung. It is often necessary to keep such a patient in a Trendelenburg position, and this usually will not interfere in any way with the position from the standpoint of the operation.

Many surgeons do not have experience in operating on a patient in the face-down position or, for one reason or another, refuse to. Frequently, this complicates the problem of anesthesia, particularly when the patient has a suppurative disease in which there is a great deal of intratracheal pus and so forth, but if the anesthetist can tilt the table into a Trendelenburg position and will recognize the fact that the diseased side is always higher than the other, the ease of aspirating the tracheobronchial tree will be greatly enhanced, and the patient will have little difficulty.

Also there is the factor mentioned by Overholt and others. They seem to believe that the emptying and the filling of the heart depend greatly on the patient's position. While there may be a certain element of truth in this, I don't believe it has been definitely proved, and it is not borne out from the clinical standpoint, because in most cases the position has little effect on the ultimate outcome of the case.

My opinion is that the position in thoracic surgery is primarily a mechanical feature and one that is important to the anesthetist, who must be able to keep the tracheobronchial tree clear and prevent atelectasis.

DR. HALE: Thank you, Dr. Effler.

What are the advantages of the large molecule solutions, such as dextran?

Dextran is a rather complex carbohydrate used during World War II, and it recently has been gaining popularity in this country. It has a molecular weight of 200,000 to 300,000. Therefore, it stays within the blood stream and draws fluids from the tissues, thereby preserving and increasing the blood volume. It has been used as a blood substitute.

Dextrose and sucrose have been used for the same purpose and are effective for short periods.

Blood itself and some of its products, plasma and albumin, are also useful for this purpose. Albumin, having a molecular weight of about 75,000, and globulin, with a molecular weight about twice that, are both satisfactory blood substitutes for this purpose.

The exact fate of dextran in the body is not entirely proved. Some of it may be metabolized as a carbohydrate. There have been a few reactions to it, chiefly pyrogenic. I think it is a little early to give a final opinion on the substance.

We now have the questions that you have been writing down and handing in, and I shall ask Dr. Ernstene to take one of those that have just been given him.

DR. ERNSTENE: **If digitalis causes decreased cardiac output, do you think that digitalized patients are poor candidates for cyclopropane, which also causes decreased cardiac output?**

I believe that the effect of both cyclopropane and digitalis upon the irritability of the heart muscle itself is more important than the effect of either preparation on the cardiac output.

It is true that in a person with a normal heart the administration of digitalis may lead to a reduction in the minute volume output of blood. On the other hand, in a patient suffering from congestive heart failure, the administration of digitalis results in an increased rather than in a decreased minute volume output of blood. Digitalis, if it is given in maximal amounts, may result in the appearance of extrasystoles or premature beats, owing to increased irritability of the heart muscle tissues.

Cyclopropane, particularly when given to elderly persons, may also result in the appearance of rather frequent premature beats.

The combined effect of the two preparations, digitalis and cyclopropane, therefore, might well be expected to result in the appearance of frequent extrasystoles in certain individuals. Generally, extrasystoles are of no clinical importance, but, if they occur at frequent intervals, the irregularity may progress to a much more serious type of disturbance, namely, ventricular tachycardia.

Because of this, I feel that a person who has had congestive heart failure, and who has had digitalis, should not receive cyclopropane as the anesthetic agent.

DR. HALE: Thank you. Dr. Taylor will you answer one of yours?

DR. TAYLOR: **What measures can be taken to compensate for the excess adrenalin secretion in pheochromocytoma when the tumor is surgically removed to avoid ensuing shock?**

Pheochromocytoma can produce persistent or intermittent hypertension and all the complications that accompany high blood pressure in human beings. It was once thought that this substance was primarily adrenalin, but more recently extracts of these tumors have been shown to contain a large percentage of a substance called noradrenalin. This induces physiologic reactions much different from those produced by adrenalin as far as the heart and circulation are concerned. If over 85 per cent of the secretion of a medullary tumor is noradrenalin, it is understandable that persistent hypertension without tachycardia, and in some cases without change in metabolic rate and blood sugar, can occur.

The presence of noradrenalin in the tumor makes it a little more difficult to control the secretions during the operation. There are various adrenolytic and sympatholytic agents now available, and many of them are in use experimentally at the present time. Those commercially available are dibenamine, Priscoline, and benzodioxane. The oldest is benzodioxane, which was marketed some time ago by the French and called F933. This drug inhibits the pressor effect of adrenalin but not the depressor action adrenalin can cause. Hence, if there is an excess amount of adrenalin, one wonders whether benzodioxane should be used. However, if given slowly as an infusion, it probably can be used safely, since the duration of its effects is short.

Priscoline inhibits the pressor action of adrenalin and prevents vasodepression to some degree; further, it has an effect on noradrenalin.

Probably the best agent is dibenamine, which may be given intravenously, acts from twelve to twenty-four hours, and has an inhibiting effect upon noradrenalin as well as adrenalin. The effect persists without constant infusion, and the agent has but slight depressor effect in its own right.

Any of these drugs may be used. It depends a lot on the condition of the patient, and one must consider whether or not one can risk inhibiting entirely the emergency, blood pressure-increasing properties of adrenalin during an operation. Probably dibenamine is the safest; the others may be used at the discretion of the physician.

DR. HALE: Dr. McCullagh.

DR. MCCULLAGH: It is only fair to mention, in connection with pheochromocytoma, that it has been recently shown in the U. S. Pharmacopeia Standard that epinephrine, or adrenalin, is in large part not adrenalin at all.

The question I have in my hand is: **What is the recommended preparation for anesthesia of a patient with hypoglycemia associated with a pancreatic tumor?**

The recommended preparation is that which will prevent the hypoglycemia typical of most tumors of islet cells. That means that a relatively high carbohydrate intake should be given orally, as close to the time of operation as possible, and that glucose should be given intravenously up to the time of, and throughout, the operation.

After removal of such a pancreatic tumor, with which there has been attendant hypoglycemia, there is normally a considerable increase in blood sugar levels, and a relative diabetes exists for a short time. There is apparently no compensatory atrophy of the remainder of the islet cells in the pancreas, such as the atrophy that occurs as a compensatory mechanism in other glands, e.g., the adrenal. One adrenal may become compensatorily atrophied to the opposite one.

Hypoglycemia occurring after such operations for pancreatic tumor can be largely disregarded. Insulin that comes from the tumor will wear itself out within four or eight hours, and the blood sugar increase will probably last a day or two, or not longer than a week or two. It is not serious, nor should it require special care.

Dr. Hale: I should like to ask the members of this panel who disagree with what another member is saying to express their opinions, as those are the things that make the panel most interesting.

Dr. Effler has a question.

Dr. Effler: I have two questions, and they are related. With your permission, I should like to answer them both at once.

What is the anesthetic and technic of choice for any thoracic operation?

I will modify the question and simply name my choice of anesthetic, because, as you know, the choice of anesthetic agent for any specialty work is always a matter of personal opinion.

Briefly, I prefer cocaine, atropine, pentothal sodium, gas, oxygen, and ether; in other words, practically the whole pharmacopeia from the standpoint of the anesthetist. That is to say, I prefer intratracheal anesthesia with pentothal sodium induction and oxygen-ether maintenance. I prefer pentothal sodium induction with various amounts of pentothal sodium after the patient has been prepared with cocaine, usually a 4 per cent solution, care being taken before the cocaine is given to see that he is well protected with atropine. Occasionally, the anesthetist uses nitrous oxide.

My experience with cyclopropane is extremely limited. It would be unfair for me to express an opinion on it. I honestly believe it is not a safe anesthetic agent in all hands, mainly because of the occurrence of cardiac complications. I would not care to engage in any arguments with any one who is an advocate of cyclopropane.

The second question is: **What is your opinion in regard to bronchoscopy directly following thoracic surgery?**

I think bronchoscopic aspiration of the trachea is the surest, most accurate way of assuring a clear airway when the patient leaves the operating room. On the other hand, I don't like it or any other completely routine procedure. I dislike being committed to the same procedure all the time. I prefer the anesthetist's routine and will give him his preference. I respect his judgment on whether the patient's secretions are sufficient to warrant bronchoscopy. If he has been energetic and has carefully observed the

patient, he knows whether the patient needs bronchoscopy, or whether simple catheter suction of the intratracheal tube will be sufficient.

I wish to say that bronchoscopy after any thoracic procedure, particularly on an older patient or one who has had a difficult operation, is not without its hazards. Anoxia and cardiac arrest are not uncommon. For that reason, Dr. Hale and his associates at the Cleveland Clinic make it a point of seeing that the patient is always prepared for postoperative bronchoscopy as he would be for any bronchoscopy. The preparation usually consists in giving atropine intravenously and then a few drops of cocaine solution through the intratracheal tube just before the tube is removed and bronchoscopy is carried out.

DR. HALE: Following Dr. Effler's lead, and not wishing to take up too much of this program, I will answer quite a few of these questions because some of them seem to be purely anesthetic. I should like to have any other member of the panel, however, speak up at any moment if he can add something or contradict something.

Concerning hyperventilation, postoperative value, contraindications, methods of hyperventilation; how soon, how many times after surgery, and equipment used.

I presume this means with carbon dioxide. It is difficult to get a patient to hyperventilate if you do not give him a strong stimulus. I think it has its place.

Some objection has been raised to the use of 5 or 10 per cent carbon dioxide in oxygen, because if the patient breathes this mixture for a few moments, he gets a good carbon dioxide effect, to be sure, but immediately afterwards goes into a period of apnea. During this time his lungs are filled with oxygen and little nitrogen; the oxygen being readily absorbed, atelectasis, the condition we are trying to avoid, may result. On this account, I know one anesthetist who prefers to administer carbon dioxide in the pure state from a catheter held above the patient's nose. Carbon dioxide, being heavier than air, will flow to the patient and still leave nitrogen in his lungs, nitrogen not being absorbed so rapidly as oxygen.

There have been other developments that take the place of this sort of thing. Of course, breathing in a bag should be mentioned. It is an old trick, and I think it is as valuable today as it has ever been.

The use of intercostal blocks or other means of preventing postoperative pain is a procedure that attempts to accomplish the same purpose, that is, it allows the patient to breathe deeply. The patient who needs this most, I believe, is the one who has had an operation on the chest or in the upper abdomen.

What type of anesthesia should be used for a patient with a large sublingual abscess with gasping cyanosis?

I believe that the preferred procedure is blind intubation (or intubation using a laryngoscope) of the trachea before any anesthesia is started. This can be readily accomplished if the pharynx and larynx are cocainized. After intubation any general anesthetic agent may be given.

Dr. Taylor: How about that 15 gage needle you are proposing to carry around in your pocket all the time in preparation for a condition like that?

Dr. Hale: Some of us believe that enough oxygen to sustain life can be delivered to a person through a 15 gage needle passed into the trachea, and on this account we have our eyes open for a suitable subject. It has been done very satisfactorily on a dog and in a case exactly like this. If the administration of an anesthetic is begun without an airway's being guaranteed, and if mucus gets into the pharynx and into the trachea and as a result intractable spasm occurs, we can administer oxygen directly into the trachea through a needle in amounts sufficient to sustain life.

Dr. Effler: I have seen two deaths from quinsy when an attempt was made to use surgical drainage, which was certainly indicated, and to give the patient an anesthetic at the same time. Some of these patients are so sick that they require no anesthetic. The only difficulty is that such a patient is so sick he is unable to expectorate the pus.

If I saw a patient with sublingual abscess and gasping cyanosis, I would treat the patient for cyanosis before treating him for the abscess producing it. If it were far enough up the neck so that a tracheotomy could be performed, I would insist on the surgeon's doing a tracheotomy and then give the anesthetic.

Dr. Hale: **Why the extreme decrease in blood pressure following pentothal sodium and curare anesthesia? This is usually after the patient has been returned to bed.**

I presume in this combination nitrous oxide and oxygen are also given, and it is conceivable that in this combination some carbon dioxide may have accumulated in the system. The patient's blood pressure would be sustained by the carbon dioxide, only to fall at the termination of the anesthesia when the carbon dioxide is also eliminated.

It is true, however, that the muscular relaxation caused by curare removes the support of many of the veins—in the extremities, particularly—and thereby allows blood to pool in them. This is the commonly accepted cause of hypotension after the use of this agent.

What anesthetic is to be preferred for the patient with asthma?

I think ether is commonly accepted as a good antibronchospastic agent and is often used on that account. I believe many other agents are satisfactory if the patient's asthma is not severe and if it is known to respond to injections of epinephrine.

What are your objections to pentothal sodium-oxygen-curare for major operations with the exception of chest surgery?

I don't believe there are any objections. I think nitrous oxide should be added to this mixture. It reduces the quantity of the other agents needed, and I have seen perfect relaxation of the abdomen when this combination has been used.

In one clinic where spinal anesthesia was always considered the method of choice for upper abdominal operations, this combination has been used many times with satisfaction.

What diagnostic tests or observations do you use to determine reaction to blood transfusion when the patient is under anesthesia?

One sign that is helpful is sudden oozing in every part of the operative field as the transfusion is given. Another is a decrease in blood pressure which persists when we would expect an arrest in the decrease or even an increase in blood pressure if compatible blood were given. Cyanosis may appear if the reaction to incompatible blood is pronounced.

Why is the administration of 100 per cent oxygen not a safe concentration to use with pentothal sodium?

I think I touched on this earlier when I mentioned the fact that at the conclusion of the operation there is enough oxygen in the lungs to carry on a while; if this is absorbed, the lungs may collapse. I think it is wise to combine nitrous oxide with the oxygen, because it is soon eliminated.

I shall now go over some of these new questions and in the meantime will ask Dr. Ernstene to take another of the questions he has.

DR. ERNSTENE: **To what extent do you feel it is indicated to give procaine intravenously in the treatment of cardiac arrhythmia?**

I believe that the intravenous use of procaine for the management of certain cardiac arrhythmias has not been completely evaluated. For the time being, however, there seem to be two principal indications: first of all, for the patient who has numerous premature beats, and in whom the irregularity is not corrected or diminished by the increased administration of oxygen; secondly, upon the occurrence of ventricular tachycardia.

The premature beats in the first instance should be of ventricular origin. The only way to be sure they are of ventricular origin is to secure an electrocardiogram. Ventricular tachycardia is also an indication for an electrocardiogram for precise diagnosis.

I am not at all sure that the intravenous administration of procaine represents the ideal treatment in either of these problems. For instance, a certain number of attacks of ventricular tachycardia can be terminated by the administration of atropine.

DR. TAYLOR: **What methods are available for maintaining nutritional requirements before and after gastric resection?**

There are many available. The ones that are most interesting and worth while mentioning here are those that enable one to maintain animals in a state of complete nutrition by parenteral administration of foods. There are emulsions of fat now being studied that can be given intravenously without inducing any serious reaction in animals.

You are all familiar with the amino acid preparations that are available. They are prepared by hydrolyzing casein and other proteins.

A combination of these amino acid mixtures, emulsified fat, and carbohydrates makes it possible to maintain in good health an animal whose

esophagus has been ligated. Nutrition is further augmented in the animal by the use of additional iron, vitamins, and other products that can be given intramuscularly. However, these measures are not yet available for routine clinical use. The amino acids are in routine use, and it has been shown that they are utilized by the body, when the liver is not diseased, to manufacture proteins needed in ordinary metabolism.

If the gastric resection is total, I think most surgeons believe that feedings may be begun a relatively short time after operation. There is no reason why such a patient can't start eating three or four days postoperatively.

Preoperatively, if the patient has extreme malnutrition and if there is any hope that the carcinoma or whatever is causing the obstruction can be removed, jejunostomy may be done and nutrition restored by tube feedings. It is interesting to know that sooner or later we should be able to maintain normal nutrition by parenteral means alone.

Dr. Effler, I think you are the only *bona fide* surgeon here. Don't surgeons start feedings within two to four days after gastric resection?

Dr. Effler: Yes, I think in most cases of gastric resection all surgeons do. I think the résumé by Dr. Taylor is excellent.

I would like to say that the feeding and nutrition of a patient prior to and after operation certainly should not be a responsibility foisted off on the anesthetist, as it is occasionally.

Another thing that I think is important: A patient who needs nutrition, particularly of the amino acid type with adequate preparation, does not require it during the time of operation. The anesthetist has his own problems, and the maintaining of a satisfactory blood volume and the giving of fluids and other agents indicated are enough without the worry about the addition of various types of amino acids and so forth. I have seen that done, and I think it is unfair both to the patient and to the anesthetist. When you are called upon to do that, stand on your constitutional rights and say you will put the patient to sleep and keep him relaxed, but that you are not going to be responsible for feeding him.

Dr. McCullagh: **What is the anesthetic of choice for a severely toxic patient with untreated hyperthyroidism who is going to have an emergency appendectomy?**

That is a tough proposition. If the question were worded to ask me what I would do, I would answer that I would consult the anesthetist. I would ask Miss Adams or I would ask Dr. Hale.

It obviously is a real problem when one meets, as rarely would occur, an emergency situation of this type or some similar type in a patient with severe untreated hyperthyroidism. Thyroid crisis certainly must be avoided, and an acute infection or trauma of any kind would produce it quickly.

The anesthesia of choice, as far as I know, would be as little anesthesia as possible, as brief as possible, and much local as possible, and sufficient preoperative medication.

What precautions should be taken to prevent thyroid crisis?

Nowadays, I should think that one would wish to put off an emergency operation, if it could possibly be done safely, until some control of hyperthyroidism could be obtained. If appendicitis develops in a person who has obvious severe hyperthyroidism, there might be instances in which the operation could be postponed safely, depending upon the situation at hand and the use of antibiotics to the best possible advantage.

The administration of iodine in almost any form and in large enough doses is still the best method of preventing or treating hyperthyroid crisis.

Certainly, one must not depend on thiourea drugs for this purpose. They have changed the whole picture of the treatment of hyperthyroidism but not in cases of crisis. They act too slowly for that. Control may not be obtained with the thioureas for some time, so iodine should be given by mouth or intravenously. It may be given as sodium or potassium iodide intravenously and the iodine administration continued for a matter of fifteen days or until such time as iodine in some form may be given orally.

The next part of this question reads: **What can we do if heat retention occurs during the course of anesthesia?**

I presume that this refers to the same patient in the same terrible state. Heat retention or sudden temperature increase in the presence of hyperthyroidism usually is the first evidence of severe crisis, and when that occurs, one really is up against it. Every type of supportive therapy at hand should be used, and the methods usually employed to reduce the temperature are those with which you are all familiar. Packing in ice and sometimes ice water enemas help decrease the temperature, but the main treatment still revolves about the management of hyperthyroidism per se.

DR. EFFLER: **Discuss the value of chest drainage postoperatively with the closure of the chest.**

Chest drainage usually consists of the use of an intercostal catheter connected with water-seal suction devised to maintain negative pressure in the pleural space. It has a real value and one not thoroughly understood in the medical profession.

First of all, as a general rule, drainage is the responsibility of the operating surgeon. I think most surgeons follow the premise that chest drainage is never used in pneumonectomy, because with the removal of a lung a large space remains that can only fill up with air or with fluid; the employment of constant drainage suction creates a great deal of negative pressure, and the mediastinum shifts to the affected side. This greatly overdistends the remaining good lung and causes serious impairment of heart function.

For the most part, I think it is safe to say that after pneumonectomy intercostal water-seal suction drainage is never used.

After other types of thoracotomy, when lung tissue remains and reexpansion is enhanced by the presence of the drainage tube, one is always

justified in using intercostal catheter suction. This is most often done after lobectomy. It is always done after a thoracic procedure in which there is the possibility of contamination, such as after esophagostomy or resection of the esophagus.

The postoperative use of intercostal suction is usually restricted to the patient who has not had total pneumonectomy and in whom there is the possibility of contamination, such as occurs in the case of an open air leak, in the presence of a great deal of contamination with the resection of an abscess, or when gastrointestinal surgery is being performed. The usual duration of drainage will be twenty-four hours or more. This depends entirely on the remaining lung tissue, the degree of expansion, and whether there is residual fluid.

DR. HALE: I have a few more questions here that I will go over briefly.

How do you prevent vomiting in a patient who has eaten shortly before an anesthetic is given?

In emergency operations, the setting of fractures, and so on, this is a very real problem, and I know that lives are lost in this country every year from just this complication.

Various methods have been used. One is the giving of small doses of apomorphine to make the patient vomit. Gastric lavage has been many times mentioned and rarely used. One of the best ways is to postpone the operation. Frequently this can be done. I know of a man in Philadelphia who has set fractures only on Tuesday mornings for the last five years. If the fracture occurs on Tuesday afternoon, it is set the following Tuesday morning, and he has never had one of these complications.

Another method is to use local anesthesia or some type of block anesthesia.

In desperation, however, many anesthetists during this coming year will be forced into giving an anesthetic to a patient with a full or nearly full stomach because a surgeon insists upon it.

What are the dangers of spraying the throat with cocaine in preparation for intubation?

Cocaine, I believe, in spite of the inferred dangers, is the safest drug for this purpose. It has been pointed out that the dangers lie in overdosage rather than in sensitivity. Sensitivity undoubtedly occurs, but if the total dose is limited to 200 mg., if the drug is given carefully and without epinephrine, and if the dose is not repeated without a suitable intervening period to guard against reaction, I believe the dangers can be definitely minimized.

Cocaine is used a great deal. I don't see how we could get along without it, and in spite of occasional drawbacks, I am sure that it will remain one of our favorite anesthetic agents.

DR. EFFLER: I am much interested in the problem of the use of cocaine as against the use of other topical agents, particularly the synthetics. I want to emphasize what Dr. Hale said. I don't see how we could get along

without cocaine. We could get along nicely, in my opinion, without the synthetic substitutes for it.

There are two types of reaction. One is due to overdosage, and the other is an allergic response. I firmly believe that the chances of having an allergic reaction are much greater with the use of synthetic preparations than with the use of cocaine itself. Every year we have patients who are "cocaine sensitive," but we have chosen to disregard the supposed allergy and have found these patients usually can tolerate cocaine anesthesia nicely for bronchoscopy or endoscopy. I believe the difficulty in the use of cocaine comes from overdosage or from too concentrated a solution. I don't think many people realize that a 4 per cent solution is just as effective as a 20 per cent solution.

We have the patient sit up on the operating table or at the side of the bed and put a basin in his hands. We tell him, "This medicine is nauseating and will make you sick to your stomach. Under no condition swallow it." The patient spits it out.

We used that procedure at Walter Reed Hospital on an undetermined number of patients and are using it at the Clinic on hundreds without any trouble from reactions. We have watched the results carefully in the hands of anesthetists and of the surgeons themselves. We have limited the amount we use and have not allowed the patient to swallow the drug.

DR. HALE: Thank you, Dr. Effler. I am in hearty accord with all you have said. One thing that should be re-emphasized, perhaps, is that cocaine should not be used with adrenalin. In spite of the fact that this prohibition is well known, there are many older laryngologists who insist on using it that way. The only safety factor lies in the fact that both drugs are powerful vasoconstrictors, so that absorption is limited.

There are two or three more questions.

Do you have any objection to using normal saline solution plus 5 per cent glucose with blood transfusion?

I think glucose should probably not be used with blood in a transfusion because there is a tendency for a film to form between the blood and the glucose. Glucose is added as a preservative to the blood that we draw from the blood bank, so it is not contraindicated from that standpoint.

Treatment for hiccough with pentothal sodium anesthesia.

I am not sure whether this means is it safe to treat hiccough with pentothal sodium anesthesia, or what should be done when hiccough occurs during pentothal sodium anesthesia.

I don't think pentothal sodium anesthesia is a good treatment for hiccough because the patient may be carried into too deep anesthesia.

The use of curare, releasing the traction on a retractor, the use of carbon dioxide, or blocking the phrenic nerves of the neck may stop hiccough. It should be pointed out that the phrenic nerves are heavily myelinated, and a 2 per cent solution of procaine or metycaine should be used for this block.

Is it permissible to add carbon dioxide to the mixture to aid intubation?

Yes, I think it is, although I believe as far as carbon dioxide on a machine is concerned, it is better not to use it. We use the patient's own carbon dioxide, as it never causes serious difficulty, and it is a rare occasion when we need the carbon dioxide in the pure state.

What do you consider the best preoperative medication for sympathectomy with a blood pressure of 280?

I think any of the agents are satisfactory. All patients requiring sympathectomy have high blood pressures. That is why they undergo the operation. So long as the patient is well compensated, I believe that the use of morphine, atropine, and one of the barbiturates is sufficient.

It is our custom to use spinal anesthesia for sympathectomy. Spinal anesthesia is contraindicated in some opinions because it causes such a pronounced decrease in blood pressure, but that is essentially what the surgeon is trying to accomplish, and we believe that in our experience it has been perfectly safe.

I would like to have Dr. Ernstene take another question.

Dr. Ernstene: **What causes twitching and unconsciousness postoperatively following cardiac arrest?**

I think these symptoms are the residual effects of anoxia upon the nerve centers in the brain. Whether they are to be permanent or transient residuals one cannot tell, except with the passing of time.

Dr. Taylor: I am going to twist this next question a little bit so I can answer it.

Should fluids—glucose or blood—be given during brain surgery, with special reference to increasing intracranial pressure?

We have been interested in the effect of various factors upon intracranial pressure. It has never been explained to any one's satisfaction why a person with malignant high blood pressure will have elevated intracranial pressure at times.

It is well known that hypertonic fluids given intravenously will reduce the intracranial pressure of animals and of some human beings to almost imperceptible levels if given long enough.

Finally, Dr. Hale and Dr. James Gardner have used a technic, developed by Dr. I. H. Page in the laboratory, of bleeding patients preoperatively to reduce the blood pressure to hypotensive levels. In this manner vascular intracranial neoplasms may be removed without excessive loss of blood. Their thought was to reduce pressure and prevent bleeding; also if blood pressure is reduced acutely, the clotting mechanism becomes more active, and it is comparatively easy to proceed with the operation. After the operation is completed, the blood pressure is restored to normal levels by retransfusion of the blood, arterially.

I would say, in general, that giving fluids by vein is not harmful during brain surgery.

DR. HALE: We have used blood letting, as Dr. Taylor pointed out, to reduce the blood pressure so that the surgical procedure might be facilitated, and so long as we have preserved what we have set up as a standard of minimal blood pressure for the patient during short periods only, with the patient in the horizontal position and well oxygenated, we haven't had any trouble. There are many things in such a procedure that can cause difficulties, and we have come close to many of them, but we believe it is a safe procedure and one indicated for a few selected conditions.

Dr. McCullagh, have you a question?

DR. MCCULLAGH: I understood Dr. Hale to say a while ago that some of these questions were almost purely anesthetic. I am glad to point out that most of them are quite stimulating.

This question reads: **What is the best management for a patient with hypoproteinemia? Hypoglycemia?**

I suppose if one wished to be facetious, one could say find the cause of the hypoproteinemia and remove it if you can. In other words, the hypoproteinemia must certainly be corrected according to cause, and it must be corrected preoperatively, if possible.

In general, the best means of correcting hypoproteinemia is to provide an adequate caloric intake and high protein feedings. Proteins that are digested are broken down and remade into proteins that can be utilized by the body.

In the hypoproteinemia of diabetes, for instance, we have satisfied ourselves that the low plasma albumin, which is common, can in most instances be brought up to normal, and sometimes it is interesting to find that we can increase the total plasma protein to a level above normal, by high protein feedings. This takes time.

The high protein feeding we use under such circumstances is a diet that consists of high protein food, as much as can be taken palatably. If it is found necessary to increase the daily protein intake to a level above that which would ordinarily be adequate—say, 120 Gm., which is a reasonably high intake—with other food, it may be accomplished by adding dried milk protein to skim milk mixtures and flavoring it suitably. A good many grams of this milk substance can be taken a day, and the protein of the diet brought up to a level above that which can actually be utilized by the body.

In acute hypoproteinemia we ordinarily have to depend upon the addition of protein intravenously, either as plasma, whole blood, or as fresh plasma.

You need not be told, I am sure, that the disadvantage in the use of blood plasma lies in what we have learned has been a rather frequent occurrence of hepatitis. It takes a large quantity of plasma or whole blood to increase the protein perceptibly, by measurement, but nevertheless a few transfusions, although the plasma albumin level does not increase, may help greatly as far as the patient's situation is concerned.

What is known about the susceptibility to anoxia of patients with diabetes?

This is an interesting question, and I am not sure that I can answer it adequately. I understand that the question is whether anoxia in any degree might be more harmful to an individual who has diabetes than to another person. I think that it is a definite possibility on two scores.

First, if the patient has diabetic acidosis, the intracellular oxidation processes are already diminished; therefore anoxia added through lack of oxygen supply would be more harmful than under ordinary conditions.

Fortunately, it is not usually necessary to operate on a patient during diabetic acidosis. The acidosis should, if at all possible, be corrected before the operation.

The other factor entails the blood supply. Because of the fact that both large and small blood vessels are affected with arteriosclerosis earlier in the course of diabetes than they would be in its absence, the blood supply to various parts of the body, including the brain, the heart, and the kidneys, particularly, is cut down. Anoxia, for this reason, is to be avoided more carefully under conditions of diabetes than otherwise.

The other question is in regard to the susceptibility to anoxia of patients with renal impairment.

That, I think, would apply particularly to the diminution of activity, the cells involved, and their blood supply. Dr. Taylor may have something more to say about that.

What is the best management for thyroidectomy in children?

I presume this means chiefly the management of anesthesia. Again I say I am not an anesthetist nor a surgeon. The problem has been resolved largely through medical treatment, because there is hardly an instance in which a patient needs to be operated on when hyperthyroidism exists. Some still are operated upon when they have hyperthyroidism, although a mild degree of hyperthyroidism in a person in good health otherwise is not a serious matter. However, if there is need to be concerned about the status of an individual, because of age, when thyroidectomy is thought to be the treatment of choice, because of the cardiovascular status, or because of the severity of the existing hyperthyroidism, the hyperthyroidism should be controlled before an operation is attempted and can be completely controlled by the proper use of propyl or methyl thiouracil.

It is usually a wise procedure to help the situation by prescribing iodine for about two weeks prior to operation.

What is the best management for an aged diabetic patient with arteriosclerosis who is admitted for emergency brain surgery?

This has very little, I should say, to do with the diabetes. Diabetic patients would be handled in the usual way. I have commented briefly on arteriosclerosis. I will pass the question on to Dr. Hale.

Dr. Hale: Probably the chief indication is to administer oxygen, regardless of the type of anesthetic that the patient receives.

I believe that our choice in this situation would be pentothal sodium with oxygen by machine. To be sure, pentothal sodium does decrease the

body's ability to metabolize oxygen, but at the same time such a patient needs only mild anesthetization. Local anesthesia can accomplish a great deal, and by giving plenty of oxygen I think we could handle the situation.

Our time is getting short, and I would like to suggest that we take the rest of these few questions and answer them briefly. If Dr. Effler will take each of the questions that he has left and if the other members of the panel will do the same, we will bring this meeting to a conclusion.

DR. EFFLER: **Are there any occasions when intubation is not necessary for intrathoracic surgery?**

Yes, I think there are a few, particularly in surgical draining of an empyema cavity or a fixed cavity with dense pleural adhesions; also when the operation will not involve extensive resection and there is no opportunity for a large amount of secretions to accumulate in the tracheobronchial tree. Then intubation, as a general rule, may be dispensed with.

What effect, if any, does suction have on the tidal volume during an open chest procedure?

I am sure that question refers to intrabronchial suction by the anesthetist. More air will be sucked out through the catheter than the patient is able to inspire through the normal process of breathing. It is well to remember that when you put the suction tube down, the suction should be left on for only a moment or two and turned off to allow the person to inspire. It may then be turned on as often as necessary. The tube need not be taken out each time, but the suction must be turned off. Otherwise the patient will immediately become cyanotic as the tidal volume diminishes practically to zero.

DR. ERNSTENE: **What kind of anesthetic agent should be given a patient who had a coronary attack three or four months ago?**

After three months the area of the heart muscle involved would be completely healed. If in the meantime the patient had no other symptoms, either coronary or myocardial failure, I think it would make little difference what anesthetic agent one employed except that cyclopropane should not be used. Of course, whatever agent were employed, one should be careful to avoid anoxia. Probably it is best not to use spinal anesthesia for such a patient, unless one is prepared to take whatever measures are necessary to prevent a considerable decrease in blood pressure.

DR. TAYLOR: This one questions why there should be a sudden drop in blood pressure after the patient is removed from the lithotomy position in which he has been for some time.

The legs have been drained of venous blood by gravity. When the position is changed, there suddenly is a great readjustment of circulation, with the blood rushing to the legs.

What would be your choice of procedure in anesthetizing an elderly patient with hypertension and bronchiectasis?

Hypertension would not influence the choice of anesthesia.

What about the use of epinephrine for hemostasis by the surgeon during general anesthesia?

There is no contraindication for it, unless too much is given.

Dr. Hale: I have a few brief answers to give you.

How often should an intratracheal tube be changed on a patient with bulbar poliomyelitis who is in a respirator?

I had better get in a word about tracheotomy now before I turn this question over to Dr. Effler, as I would like his opinion on this. I think it is recognized by a good many persons that tracheotomy is indicated early in a patient with bulbar poliomyelitis and should be done without delay. If an intratracheal tube is used, it should be changed once every twenty-four hours.

Dr. Effler: I have had no experience with a patient with bulbar poliomyelitis on whom intratracheal intubation has been done, but it seems to me the time for changing the tube should be reckoned in hours, and certainly not in weeks.

Dr. Hale: **What do you consider the most desirable dosage for spinal anesthesia?**

For procaine, I would say 150 mg.; for pontocaine 10 mg.; and for nupercaine, 5 to 6 mg. So many factors enter into it, that it is difficult to answer.

Differentiate between the use of serum albumin with decrease of blood pressure and its use with increased blood pressure during craniotomy.

If albumin is used to correct decreased blood pressure, it serves as a blood substitute for building up the blood volume. During craniotomy it reduces brain edema by drawing fluid into the blood vessels.

What is your treatment for spinal headache?

I believe we never have any spinal headaches, but the surgeons insist that we do. One of the best treatments I am certain must be instituted preoperatively. I recall a surgical ward at the Naval Hospital where the word was not permitted to be used by nurses or corpsmen or medical officers, and spinal headache was practically unknown.

We treat the patient with spinal headache by giving naotin, which is one of the nicotinic acid preparations, intravenously. The patient reacts with a prickly sensation, which takes his mind off his headache. We then give half normal saline solution, which allows fluid to go into the dural sac, which is redistended.

What treatment should be given for bronchial spasm?

That means during anesthesia, I presume. Ether has been given rectally for bronchial spasm. Atropine has been used. Small amounts of epinephrine may be given if there is no contraindication to it, and also aminophylline.

I think it is important that the patient get plenty of oxygen so that what breathing he does is beneficial.

Do you feel that intratracheal intubation is a must in major abdominal surgery when pentothal sodium with curare is the anesthetic?

No, I don't think it is necessary; in many cases no artificial airway is needed. Sometimes gentle pressure on the bag will carry out artificial respiration satisfactorily.

Do you advocate the use of 20 per cent procaine, 1 or 2 cc., in intravenous solution in cardiac surgery?

No. A 20 per cent solution is undoubtedly too strong. If we are going to use procaine intravenously, we can accomplish the same result by using a more dilute solution. I believe the same is true of pentothal sodium. It should be used in fairly dilute solution. We can anesthetize a patient as rapidly with a 2½ per cent as with a 10 per cent solution of pentothal sodium, and the weaker solution is much less irritating to the tissues if it gets outside the vein.

Is pituitrin contraindicated with cyclopropane?

Yes. Pituitrin is a constrictor of the coronary vessels and may add to the toxic effects of cyclopropane on the heart.

Are there any other comments which the members of the panel feel inclined to make at this time?

If not, I will turn the meeting back to Miss Adams. Thank you. (Applause)

CHAIRMAN ADAMS: I am sure we are very grateful for the forum this afternoon. I think this will finish the meeting.

My Gift

To the Agatha Hodgins Educational *Loan* Fund

I hereby donate $_____to the Agatha Hodgins Educational Fund, which is "designed to extend financial assistance to graduate nurse anesthetists in obtaining further education and training to become qualified instructors, or to instructors who desire and need additional training to become better qualified."

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NOTES

When anesthetists get together, they talk about anesthesia. They talk about gadgets, special technics, and interesting cases. This section of NOTES was originated so that anesthetists could exchange ideas in writing as they do in conversation. Send in your contribution now. Other anesthetists will be helped by it.

"Butterfly."—A device that is useful in observing the respiratory exchange is the so-called "butterfly." A small piece of Kleenex is atached to a strip of adhesive or Scotch Tape, which in turn is at-

tached to the tip of the nose and the chin. Inhalations and exhalations cause the paper to flutter, thus giving visual check of the respiratory movements. By cutting the Kleenex into strips, very delicate respirations may be observed.[1]

Abuse of Curare.—Curare and the curariform drugs have been so universally successful in conjunction with anesthesia that the dangers are often overlooked, and their clinical use is abused. Probably the most common abuse of curare is its administration to a patient when proper means of respiratory control are not available. Since very small doses of curare may, in an occasional instance, produce cessation of respiration, it is imperative that a machine suitable for controlled breathing be immediately accessible before this potent drug is administered. The most effective means of combating the overeffect of curare is artificial respiration, i. e., intermittent positive pressure until spontaneous respiration takes place.

The rapid injection of curare may, and often does, produce apnea. Sudden and fatal cardiovascular collapse may follow rapid intravenous injection of curare. Therefore cautious and controlled administration must be observed in order to avoid this all too common abuse. It is of the utmost importance that, respiratory paralysis once having been produced, the patient be helped until such time as not only intercostal muscle activity but also full diaphragmatic activity has returned. Otherwise, the patient may die of atelectasis.

The use of curare is abused when it is given before an adequate plane of anesthesia for operative intervention has been established. Since muscular relaxation attendant upon the effect of curare may disguise the planes of anesthesia as ordinarily seen, it is important that the anesthetist be assured that adequate anesthesia does exist. It has also been noted that, with inadequate anesthesia and the administration of an insufficient amount of curare to

1. Lundy, John S.: *Clinical Anesthesia* (Philadelphia: W. B. Saunders Co., 1942).

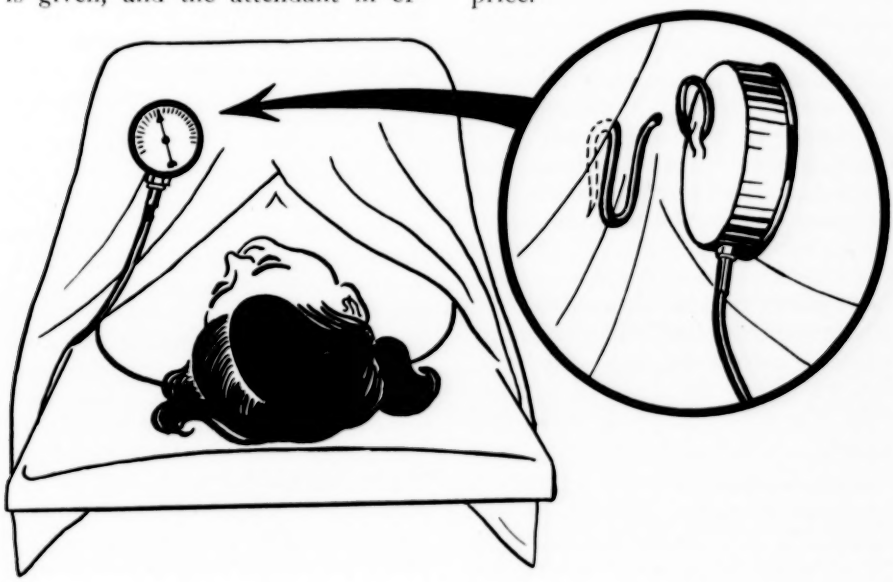
cause complete paralysis of the skeletal muscles, there is danger of the occurrence of respiratory spasms. To depend upon curare to perform any of the functions of an anesthetic other than relaxation is to use it improperly.

Careless administration of curare after an initial dose may result in cumulative action that will work to the detriment of the patient. When the drug is administered over a period of time, in divided doses, less curare will be needed with each succeeding dose. This is true also if the first dose is administered intravenously and the succeeding doses are given intramuscularly. With certain anesthetic agents, the dosage of curare must be varied. For instance, with ether anesthesia one third of the dose of curare that is given with cyclopropane will produce a comparable result.

Probably one of the most serious abuses of the use of curare is that of administering it without assurance of an adequate airway. Obstruction to respiration from any cause may be disguised if curare is given, and the attendant ill effects of obstruction will occur without the usual warning signs.

In addition to the precautions indicated by age, weight, the conditions of operation, and the need for curare, it is always well to remember that the anesthetist must assume, in addition to other responsibilities, the responsibility for a respiratory mechanism deprived of its ability to provide adequate ventilation for the patient.—EXIRE O'DAY, R.N., Chicago.

DRAPERY HOOK.—A drapery hook has been found useful for attaching a sphygmomanometer dial to the draping sheet or a pillow. This device has value when the gas machine is not equipped with a hook, or when the gas machine must be removed from the operating room. In addition to this use for the drapery hook, it may provide a means for attaching other small objects, such as forceps, to draperies near the head of the table where they are accessible to the anesthetist. The hooks may be purchased at any dime-store for a nominal price.



LEGISLATION

Emanuel Hayt, L.L.B.*

A Case of Hasty Diagnosis.[1]—The patient consulted a specialist in cancer surgery, who advised the removal of an enlarged lymph node in her right armpit for examination. Assisted by the attending doctor, the specialist excised the node and sent it to the hospital laboratory for examination. While the patient was still under anesthesia, the head nurse announced that the laboratory report was "carcinoma." The patient's right breast was then removed under the direction of the specialist, who was assisted by the other physician. At the completion of the operation, the laboratory technician came back with a report indicating that she had made a mistake.

A malpractice action was brought by the patient against the specialist and all those involved in the operation. She alleged that her breast was removed without her consent. The court held that if the removal of the breast was due to negligent haste, it was not the fault of the physician who merely acted as the assistant. All of the decisions were made by the specialist in charge, and if there was negligent haste, the fault could not be charged to the physician who had no connection with the wrong diagnosis that was brought from the hospital laboratory. The specialist had died. Under the circumstances, the patient was unable to collect.

The Anesthetist Would Have Been Liable, Although Not the Hospital.[2]—The New Jersey Superior Court on December 14, 1949, affirmed the dismissal of the case of a child, aged 4, who had been burned by a hot water bag in a charitable hospital, on the ground that charitable institutions are immune from liability to patients.

At the hospital the child underwent a tonsillectomy. He was then taken to the children's ward and placed by the anesthetist in a bed and under its covers. The nurse anesthetist neglected to remove a hot water bag that had been put in the bed for the purpose of warming it as a matter of hospital routine. The child received severe burns, which required treatment for some time and left a permanent scar.

The action was instituted by the child and his father against the hospital and the anesthetist, the claim being that the injury resulted from the negligence of the defendants. Service of process on the nurse anesthetist was never obtained, as she was a nonresident of the State at the time the suit was commenced.

At the close of the testimony at the trial, the court dismissed the action. It was urged by the patient's lawyer that the ruling of immunity should not be applied where there is gross negligence or administrative negligence. The court held that there was no such negligence and no previous case that would recognize such an exception. The fact that the hospital carried indemnity insurance did not affect the hospital's immunity.

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*Counsel for A.A.N.A.

1. Valdez v. Hankins, California District, Court of Appeal, Second District, Division One, November 14, 1949. 17 CCH Negligence Cases 630.

2. Woods, etc., et al., v. Overlook Hospital Association, 17 CCH, Negligence Cases 826.

THE NEWS

LARGE ATTENDANCE FOR SEVENTEENTH

Early requests for applications for hotel reservations indicate a large attendance may be expected for the A. A. N. A. Seventeenth Annual Meeting in Atlantic City, September 18 through 21. Applications may be obtained by writing to the A.A.N.A. Executive Office, 22 East Division St., Chicago 10, Ill. On page 129 of this JOURNAL is a form that members should use in obtaining applications.

Mildred Schulz, United States Marine Hospital, Staten Island, N. Y., has been appointed chairman of the Program Committee to replace Ann Butterworth. Tentative arrangements are being made for an outstanding clinical program and for featured events to provide entertainment as well as education for the attending members.

The business session is scheduled for Tuesday morning, at which time the reports of officers and committee chairmen will be presented and new officers and trustees elected. At the annual banquet on the evening of September 20, the Association's Award of Appreciation will be presented.

A.A.N.A. headquarters will be at the Ritz-Carlton Hotel.

A. N. A. BIENNIAL IN SAN FRANCISCO

Eight thousand nurses representing 200,000 professional nurses in the United States, Hawaii, and Puerto Rico are expected to attend the Sixteenth Biennial Nursing Convention in San Francisco, May 7-12. Scheduled for consideration is a proposed Code for Professional Nurses, which, according to Ella Best, chairman of the convention committee, will "spell out the ethical implications of economic and social forces confronting nurses today."

Other matters to be brought before the delegates include: means of filling expanded post-war nursing needs of the American public; full participation for Negro nurses in professional activities and membership in nursing organizations; and the role of collective action in determining the economic status of nurses.

MARTHA JACKSON PRESIDENT OF SOUTHEASTERN

Martha Jackson, Tampa, Fla., was elected president of the Southeastern Assembly of Nurse Anesthetists in St. Petersburg at the assembly's annual meeting, April 5-7. Flora Burg, Miami, was elected secretary-treasurer. Presiding officers for the meeting were Anne

Beddow, out-going president, Maude Yonce, Matie L. Lawrence, and Ellen McMahon.

Speakers and subjects included: Florence McQuillen, executive director of the A. A. N. A., "Your Organization, State and National"; Matie L. Lawrence, "Sodium Pentothal-Curare for Tonsillectomies"; Dr. Robert G. McNeil, "The Value of Postoperative Orders in Relation to Brain Surgery and Anesthesia"; Dr. John R. Lewis, Jr., "Possibilities of Plastic Surgery"; Dr. Glace Bittendender, "Intratracheal Anesthesia."

TEXAS PRESIDENT RE-ELECTED

Laura Hoffman was re-elected president of the Texas Association of Nurse Anesthetists at the annual meeting held in Galveston, March 7-9, 1950. Other officers for the coming year are: Elsie A. Smith, vice president; Lois Childress, secretary-treasurer; Virginia Futch, Pearl Weaver, and Allie Mae Fruge, trustees.

At this meeting a budget for the state newsletter *T-ANA* was set up to cover mimeographing and mailing of four issues.

In 1951 the state association will meet April 24-26 in San Antonio.

WESTERN STATES CONVENTION

The Western States Convention of Nurse Anesthetists met with the Western Hospitals Convention in Seattle on April 24-27, 1950. Ruth Walther, chairman of the section, presided at the opening session at which Stimpson Bullett, Seattle attorney, spoke on the United Nations. Other speakers for the four day meeting were Myra Van Arsdale, president of the A. A. N. A., Atelio Pricco, Charlotte Dowler, and Dr. Daniel Moore, all of Seattle, and Josephine Bunch, of Portland.

Six of thirteen nurse anesthetists who were honored at a meeting of the Wisconsin association in Madison on February 11. All have been in the field twenty-five years or longer: (left to right) Jessie Opdale, Mabel Johnson, Myrtle Hepp, Mary Donovan, and Sigrid Esval.

Officers of the Alabama association for 1950: (left to right) Anna Traber, trustee; Mary Blande Parks, treasurer; Nathalie Davis, trustee; Anne Beddow, president; Frances Bishop, vice president; Cubelle Gardner, secretary.

Clinics were held on Tuesday morning, and a tea and tour of Seattle were features of the Wednesday afternoon program.

The program was arranged by Nora Dell, Seattle. Vice chairman of the assembly was Olivia Byre.

QUALIFYING EXAMINATION

On May 8 nearly 300 candidates for membership in the A. A. N. A. will take the eleventh qualifying examination in thirty-three cities throughout the United States. For the first time, an examination will be taken by a candidate in Japan. Other points of examination abroad are Alaska and Hawaii.

Candidates will be notified of their success or failure within ninety days after the date of examination.

OHIO ELECTS OFFICERS

At the annual meeting of the Ohio Association of Nurse Anesthetists in Columbus, March 22-23, Marcia Williams was elected president for the coming year. Other officers installed were: Charlotte Turner, 1st vice president; Dorothy De Frances, 2nd vice president; Rose Richardson, secretary-treasurer; Daisy Parker, trustee.

RESOLUTIONS CONDEMNING COMPULSORY HEALTH INSURANCE

At winter and early spring meetings of the state associations, the matter of resolutions condemning compulsory health insurance was presented to the members. To date, the following associations have gone on record as opposing legislation designed to make health insurance compulsory: Alabama, Illinois, Massachusetts, North Dakota, Tennessee, Texas, Utah, and the Southeastern Assembly of Nurse Anesthetists.

DEATHS

Mary F. Sentner, nurse anesthetist at Aspinwall Hospital, Aspinwall, Pa., was killed in an auto-

Calendar of Coming Events

May 2-4	Tri-State Assembly of Nurse Anesthetists, Chicago
May 11-12	Carolina's-Virginia's Assembly of Nurse Anesthetists, Charleston, S. C.
May 17-19	Upper Midwest Assembly of Nurse Anesthetists, Minneapolis
May 24-26	Middle Atlantic Assembly of Nurse Anesthetists, Buffalo
Sept. 18-21	Seventeenth Annual Meeting, American Association of Nurse Anesthetists, Atlantic City

mobile accident on April 6, 1950. She was a graduate of the Misericordia Hospital School of Nursing, Philadelphia, and of the Graduate Hospital, University of Pennsylvania, School of Anesthesia. During World War II, she served for eighteen months in the Southwest Pacific.

Amelia Mayer, nurse anesthetist at Shadyside Hospital, Pittsburgh, for thirty-five years, died on April 2, 1950. She was a graduate of St. Francis School of Nursing, Pittsburgh, and of the Grace Hospital School of Anesthesia in Detroit.

Mrs. Margaret Helen Gordner, anesthetist at the Geisinger Hospital, Danville, Pa., for ten years, died in March 1950. She was a graduate of the Hurley Hospital School of Nursing, Flint, Mich., and of the University of Michigan Hospital School of Anesthesia, Ann Arbor.

Julia Filla, a member of the Minnesota association, died on March 16, 1950, of cerebral hemorrhage. She was a graduate of the Winona General Hospital School of Nursing, Winona, Minn., and of the University Hospital School of Anesthesia, Minneapolis.

Agnes Grillet, for many years anesthetist at Hotel Dieu, New Orleans, died on January 29, 1950. She received her training as a nurse at St. Margaret's Hospital, Montgomery, Ala., and was a graduate of the St. Joseph's Hospital School of Anesthesia, Chicago.

Officers of the Tennessee association for 1950: (left) Ruthie Hawne, secretary, and (right) Lucile K. Hill, president.

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ABSTRACTS

Burnett, C. H.; Bloomberg, Esther L.; Ahortz, Gerald; Compton, D. W., and Beecher, H. K.: A comparison of the effects of ether and cyclopropane anesthesia on the renal function of man. J. Pharmacol. & Exper. Therap. **96**:380-387, Aug. 1949.

"We report here the results of measurement of renal function just prior to and during ether and cyclopropane anesthesia in normal humans. No surgery was undertaken before completing the observations. . . . Mannitol and sodium para-aminohippurate were used for measurement of glomerular filtration rate and effective renal plasma flow, respectively. . . . An attempt was made to exclude complicating factors that produce a divergence from homeostasis except those caused by the anesthetic agent itself. The patients chosen were those in whom elective surgical procedures were anticipated. They were in good clinical condition, and had normal kidney function by the usual rough clinical criteria. . . . Usually, no premedication had been given, and some patients were rather apprehensive. Hence, the effect of excitement on renal function cannot be excluded during the pre-anesthetic stage. During induction and until the patient had reached the first to second plane (Guedel) of the third stage of anesthesia, clearance measurements were interrupted. Collections were then started again and continued at approximately ten-minute intervals for an additional 23 to 74 minutes. Anesthesia was maintained insofar as possible at these same levels throughout the period of observation. Eight patients received ether, and seven received cyclopropane....

"Judging from the observations reported here, ether is preferable to cyclopropane in patients if shock is present or anticipated during operation. . . . In seven of the eight patients who received ether, and in all who received cyclopropane, the average glomerular filtration rate and effective renal plasma flow fell during anesthesia. The average filtration fraction rose in each patient except in one, who also showed no significant changes in mannitol or para-amino-hippurate clearances. Average urine flow fell in both groups, accompanied by a rise in the average U/P ratio. Individual variations in all of these measurements were observed. The changes in kidney function observed during ether and cyclopropane anesthesia were qualitatively similar; quantitatively, they were greater with cyclopropane, and somewhat less subject to individual variation. Thus, average glomerular filtration rate fell 21 per cent with ether, and 31 per cent with cyclopropane; average effective renal plasma flow fell 39 per cent with ether and 52 per cent with cyclopropane; average filtration fraction rose 25 per cent with ether and 35 per cent with cyclopropane."

Berger, F. M.: Spinal cord depressant drugs. J. Pharm. & Exper. Therap. **96**:243-278, Aug. 1949.

"The pharmacology of drugs possessing a depressant action on the spinal cord received little attention in the past. The discovery of useful applications of curare in medicine and surgery directed attention to other substances capable of producing paralysis. The purpose of the present article is to summarize the pharmacological properties of various substances producing paraly-

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sis of skeletal muscles by a depressant action on the spinal cord. Because of the lack of suitable laboratory methods for the evaluation of the therapeutic potentialities of these substances, the clinical results obtained with some of them have also been briefly reviewed. The actions of local anesthetic drugs on the spinal cord were specifically omitted. . . . Most substances possessing a depressant effect on the spinal cord have the important common property of producing paralysis of the ascending type. Tri-o-cresyl phosphate, dithiobiuret, benzimidazole, myanesin and glyketal depress the lower segments of the cord in small doses. With larger doses, higher segments of the cord and the midbrain are also affected in an ascending order. The anesthetics differ from the spinal depressants by first depressing higher levels of the central nervous system and by producing paralysis at lower levels only after large doses. The vital functions of the medulla are spared by both the anesthetics and spinal depressants and are affected after toxic doses only. Whether the spinal depressant action of these agents is direct or is secondary to their effects on certain structures of the midbrain is not known. . . . The available agents producing depressant effects on the spinal cord may be arbitrarily classified into 4 groups: 1) agents producing irreversible paralysis by damaging the anterior horn cells, such as tri-o-cresyl phosphate; 2) agents producing reversible paralysis on chronic administration, such as dithiobiuret; 3) agents causing transient paralysis and possessing a selective depressant action on the interneurons, such as the benzimidazole, the glycerol ethers, the 2, 2-alkyl-4-hydroxymethyl-1, 3-dioxolanes, and apo-*B*-erythroidine; and 4) agents which depress interneurons but do not cause paralysis, such as parpanit. It is of interest that four chemically different classes of compounds produce similar and highly selective effects on interneurons and postural reflexes. There is no indication that these compounds affect transmission of impulses mediated by acetylcholine. Their mode of action is unknown and when uncovered may bring to light new aspects concerning the transmission of impulses in the central nervous system.

"The four families of chemicals blocking interneuronal transmission differ from each other in the intensity of this action and in their antagonism to the effects of strychnine, which to some extent is possessed by all. Glyketal, the most potent interneuronal blocking agent, possesses the weakest anti-strychnine action. Benzimidazole has the strongest antagonistic action to strychnine and the weakest action on interneurons. Myanesin possesses both interneuronal blocking action and anti-strychnine properties to a marked degree. Apo-*B*-erythroidine has a blocking action on interneurons of an order similar to myanesin. Its antagonistic action to strychnine has not yet been investigated. Spinal depressants and interneuronal blocking agents are of potential therapeutic value in the treatment of muscle spasm, spasticity, tremor and involuntary movement. They may also be of value for the production of muscular relaxation during anesthesia. Up to the present, only myanesin has received adequate clinical trials. Although it has produced remarkable effects in numerous cases, the value of the drug is limited. It is unsuitable for routine intravenous use because of the low solubility and the hemolytic action of the drug.

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The intravenous use of dilute solutions of myanesin during emergencies such as tetanus, strychnine poisoning or status epilepticus may be justified, but intramuscular administration which should be equally effective and less dangerous would be preferable. The oral use of myanesin is impeded by the low potency and rapid inactivation of the drug. In the past, the activity and potential clinical usefulness of spinal depressants has been evaluated in terms of their paralysing potency. This criterion of activity is unsuitable because it is not intended to use these agents clinically for the production of paralysis. It appears probable that the interneuronal blocking action or the antagonistic action to strychnine would be a better criterion for assessing the clinical potentialities of these drugs; however, it is not known which of these two unrelated properties is the more important one. This question may be answered by clinical trials of benzimidazole and glyketal, the results of which may indicate new approaches toward the development of more effective agents of this type."

A. A. N. A.

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CURARE

(*Continued from page* 83)

has been outlined, and certain curare preparations have been discussed. New curare preparations will undoubtedly continue to appear, and the importance of a thorough understanding of a new drug cannot be overemphasized. Some of the complications that may be encountered have been pointed out and the treatment considered.

When the nurse anesthetist begins the use of a drug or technic with which she is unfamiliar, she should obtain all the information possible, secure the co-operation of the surgeon, and then proceed with alertness and caution. She should not choose a difficult case on which to test the method, but rather choose an easy one that she may learn her way before she has to deal with complications.

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BOOK REVIEWS

RESUSCITATION AND ANESTHESIA FOR WOUNDED MEN. By Henry K. Beecher, A.M., M.D.; Henry I. Dorr Professor of Research in Anesthesia, Harvard University, Chief, Department of Anesthesia, the Massachusetts General Hospital. Cloth. 161 pages, 58 illustrations. Springfield, Ill.: Charles C Thomas, Publisher, 1949.

The material for this book was obtained during military service. Much valuable information for anesthetists in civilian practice is presented. Comparison of the treatment and the results after World War I and World War II, as well as in the early part of World War II and late in the war, shows the improvement in the care of wounded men.

All factors affecting such care are considered in detail; emotional problems, pain, the nature of the wound, shock, and methods of evacuation.

The physiologic effects of wounds are presented in detail, with many graphs and tables to summarize the data, as well as the care of wounded men, with discussion of the lessons learned during actual combat experience.

A series of revealing tables gives sample data pertaining to anesthesia service for battle casualties. The problems of supply, personnel, choice of agent, and administration are among the subjects discussed.

Twenty-eight pictures help the reader visualize the circumstances under which the text was developed. Thirty-two tables add to the wealth of information. Comments and bibliographic references are contained in footnotes. Indexed.

FUNDAMENTAL CONSIDERATIONS IN ANESTHESIA. By Charles L. Burstein, M.D., Chief, Department of Anesthesiology, Hospital for Special Surgery, Attending Consultant in Anesthesia, Veterans Administration Hospital, Bronx, N. Y., Assistant Visiting Anesthetist, Bellevue Hospital, Instructor in Anesthesia, New York University College of Medicine. Cloth. 153 pages, 64 illustrations. New York: The Macmillan Co., 1949. $4.00.

The author has presented studies that have resulted from both his experimental and his clinical experience. Physiologic and pharmacologic considerations of some of the more important phases of anesthesia are included. The author claims that all of the material is new. The autonomic nervous system, respiration, laryngeal spasm, shock, circulatory disturbances, vagal and other parasympathetic reactions, and gastrointestinal autonomic reactions are among the subjects discussed. The book is indexed and has bibliographic references following each chapter. Line drawings, graphs, and pictures supplement the text.

MITCHELL'S PEDIATRICS AND PEDIATRIC NURSING. By Robert A. Lyon, M.D., Associate Professor of Pediatrics, University of Cincinnati, Assistant Medical Director, Cincinnati Children's hospital, and Elgie M. Wallinger, R.N., B.S., M.A., Director of Nursing, Columbus Children's Hospital, Ohio; with a chapter on Preventive Pediatrics by Rena M. Gazeway, R.N., B.S., M.A. Ed. 3. Cloth. 590 pages, 97 illustrations. Philadelphia: W. B. Saunders Co., 1949.

In this textbook on pediatric nursing, the nurse anesthetist will find many valuable clues for the handling of children during anesthesia. Chapters on prematurity and attendant difficulties, the abnormalities of the newborn, respiratory and circulatory systems of children, and diseases of the nervous system all contain valuable information for the anesthetist. No specific reference is made to anesthesia for children but the book is, nevertheless, well worth the anesthetist's

time. An appendix contains a table of average weights and heights and blood values for children. Many of the nursing procedures may be adapted to nursing care in relation to anesthesia. Indexed.

Gynecology and Gynecologic Nursing. By Norman F. Miller, M.D., Professor of Obstetrics and Gynecology, University of Michigan Medical School, and Betty Hyde, R.N., Supervisor of the Gynecologic Wards, University of Michigan Medical School; with chapters on the Gynecology Operating Room by Molly Kowal, R.N., formerly Operating Room Supervisor, University of Michigan Medical School, and on the Psychosomatic Approach in Gynecology by Sprague Gardiner, M.D., Assistant in Obstetrics and Gynecology, University of Indiana Medical School. Ed. 2. Cloth. 485 pages, 240 illustrations. Philadelphia: W. B. Saunders Co., 1949.

This textbook for nurses emphasizes fundamentals concerning diseases of women and nursing care involved in treatment. The authors have given the reasons for, as well as the procedures in, each treatment. In the chapter on the operating room are included the preparation of the anesthesia room and the procedures of the operating team in assisting the anesthetist. An excellent chapter on postoperative care of gynecologic patients and a discussion of postoperative complications will be of interest to the anesthetist. The chapter on the psychosomatic approach in gynecology will also be valuable to anesthetists in understanding patients with psychosomatic problems. An appendix gives an outline for nursing procedures. An index is included.

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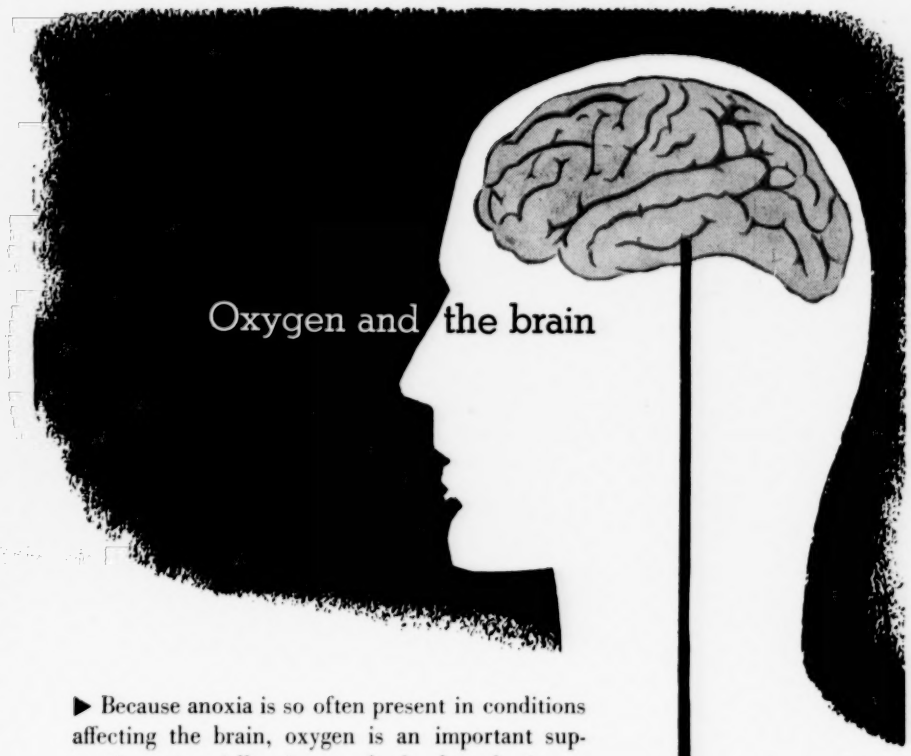

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